"COMPARISON OF EFFICACY OF 0.75% ROPIVACAINE ALONG WITH COMBINATION OF 0.75% ROPIVACAINE AND DEXMEDETOMIDINE IN AXILLARY BRACHIAL PLEXUS BLOCK FOR FOREARM AND HAND SURGERIES"

Dissertation submitted in partial fulfillment of

M.D. DEGREE EXAMINATION

M.D. ANAESTHESIOLOGY- BRANCH X

CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU.

APRIL 2015

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"COMPARISON OF EFFICACY OF 0.75% ROPIVACAINE WITH COMBINATION OF 0.75% ROPIVACAINE AND DEXMEDETOMIDINE IN AXILLARY BRACHIAL PLEXUS BLOCK FOR FOREARM AND HAND SURGERIES"** submitted by Dr.Arun. S in partial fulfillment for the award of the degree of Doctor of Medicine in Anaesthesiology by the Tamilnadu Dr.M.G.R , Medical University,Chennai is a bonafide record of the work done by him in the CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU, during the academic year 2012-2015.

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DECLARATION

I, **Dr. Arun.S**, solemnly declare that the dissertation **"COMPARISON OF EFFICACY OF 0.75% ROPIVACAINE ALONG WITH COMBINATION OF 0.75% ROPIVACAINE AND DEXMEDETOMIDINE IN AXILLARY BRACHIAL PLEXUS BLOCK FOR FOREARM AND HAND SURGERIES"** is a bonafide work done by me in the Department of Anaesthesiology, Chengalpattu Medical College & Hospital,Chengalpattu, after getting approval from the Ethical committee under the able guidance of **Prof.Dr. SUGANTHARAJ ANURADHA M.D.,D.A.,** Professor HOD, Department of Anaesthesiology, Chengalpattu Medical College, Chengalpattu.

Place : Chengalpattu

Date : **(Dr.S.Arun)**

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APPROVAL OF ETHICAL

To

Dr.S.Arun
Post Graduate
Dept of Anaesthesia

Dear Dr.

The Institutional Ethical Committee of Chengalp
and discussed your application to conduct the cl

COMPARISON OF ROPIVACAINE & ROPIVACAIN
NEUROSTIMULATION GUIDED BRACHIAL PLEXU

On 13.11.2013

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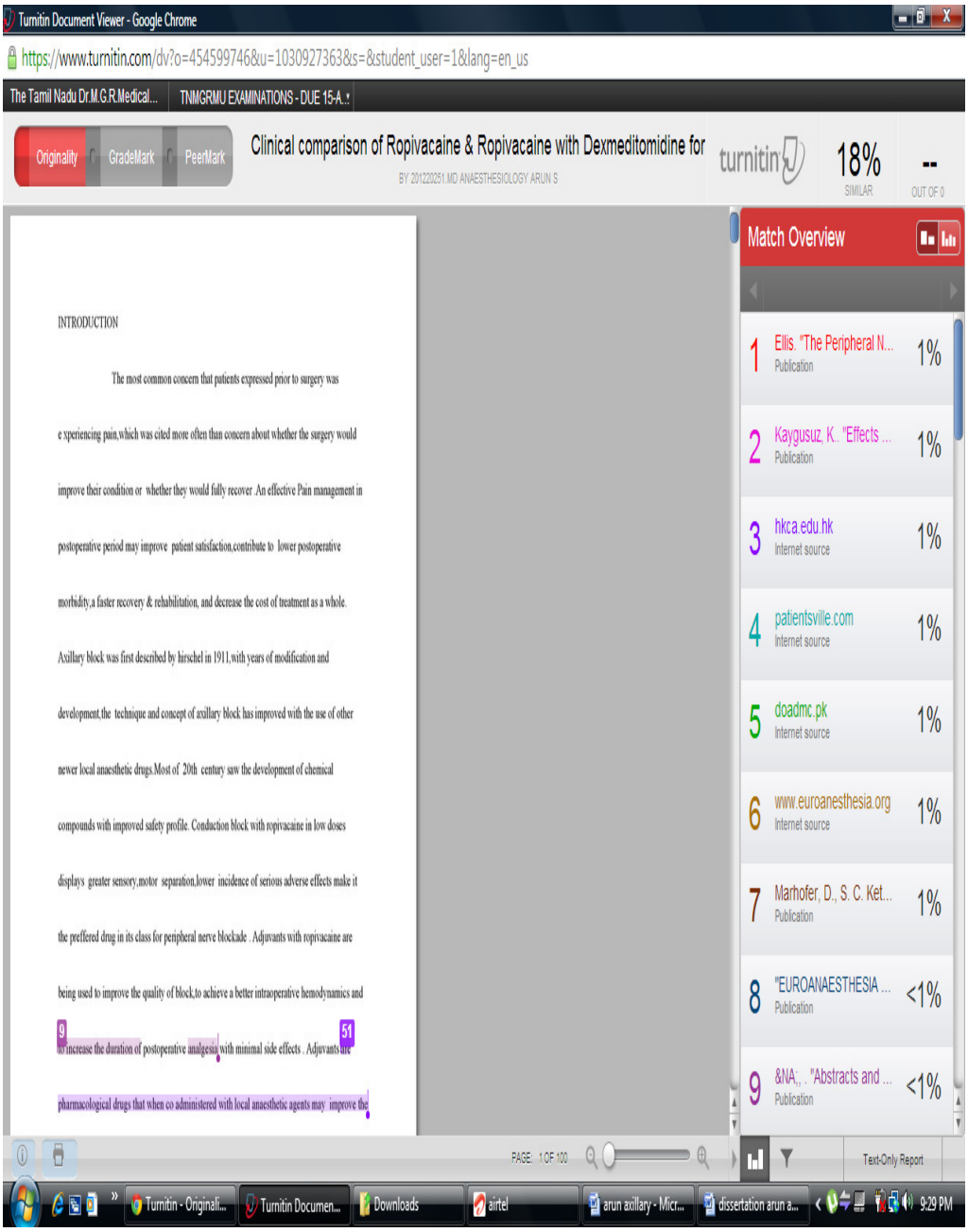
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INTRODUCTION
The most common concern that patients expressed prior to surgery was
e xperiencing pain,which was cited more often than concern about whether the surgery would
improve their condition or whether they would fully recover .An effective Pain management in
postoperative period may improve patient satisfaction,contribute to lower postoperative
morbidity,a faster recovery & rehabilitation, and decrease the cost of treatment as a whole.
Axillary block was first described by hirschel in 1911,with years of modification and
development,the technique and concept of axillary block has improved with the use of other
newer local anaesthetic drugs.Most of 20th century saw the development of chemical
compounds with improved safety profile. Conduction block with ropivacaine in low doses
displays greater sensory,motor separation,lower incidence of serious adverse effects make it
the preffered drug in its class for peripheral nerve blockade . Adjuvants with ropivacaine are
being used to improve the quality of block,to achieve a better intraoperative hemodynamics and
to increase the duration of postoperative analgesia with minimal side effects . Adjuvants are
pharmacological drugs that when co administered with local anaesthetic agents may improve the
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"COMPARISON OF EFFICACY OF 0.75% ROPIVACAINE ALONG WITH COMBINATION OF 0.75% ROPIVACAINE AND DEXMEDETOMIDINE IN AXILLARY BRACHIAL PLEXUS BLOCK FOR FOREARM AND HAND SURGERIES "

ABSTRACT:

Background: Dexmedetomidine is an selective alpha-2-receptor agonist. Several studies have described effects of dexmedetomidine on peripheral nerve blocks which might be used as an additive to local anaesthetics for various regional anaesthetic techniques. We therefore designed this prospective, double-blinded, controlled volunteer study to investigate the effects of dexmedetomidine as an adjuvant to Ropivacaine on peripheral nerve block.

Objective: In this study, we aimed to investigate the effects of adding dexmedetomidine to 0.75% Ropivacaine for an axillary brachial plexus block with respect to onset and duration of sensory and motor block and duration of analgesia.

Methods: 60 patients of American Society of Anesthesiologists physical status I/II scheduled to undergo forearm and hand surgery, in which an axillary block was used, were randomly divided into 2(30 each) groups:
Group R patients - 25 ml Ropivacaine 0.75% plus 1 ml of normal saline.
Group D patients -25 ml Ropivacaine 0.75% and 1 mL dexmedetomidine (50 µg)
Demographic data, mean arterial pressure ,heart rate , peripheral oxygen saturation, sensory and motor block onset times and block durations, time to first analgesic use,total analgesic need, postoperative visual analogue scale data, and side effects were recorded for each patient.

Results: Sensory block onset time was shorter in group D ($P < 0.05$) . Sensory block duration and time to first analgesic use were significantly longer in group D ($P < 0.05$). No statistically significant changes in intraoperative MAP and HR, spo2 among two groups. Bradycardia, hypotension, hypoxemia, nausea,vomiting, and any other side effects were not seen in any patients.

Conclusions: It was concluded in our study that adding dexmedetomidine to axillary brachial plexus block shortens sensory block onset time, increases the sensory block duration and time to first analgesic use, and decreases total analgesic use with no side effects.

Key word: Dexmedetomidine, Ropivacaine, Brachial Plexus Block

INTRODUCTION

An effective Pain management in postoperative period may improve patient satisfaction, contribute to lower postoperative morbidity, a faster recovery & rehabilitation, and decrease the cost of treatment as a whole.

Axillary block was first described by Hirschel in 1911,with years of modification and development, the technique and concept of axillary block has improved with the use of other newer local anaesthetic drugs. Most of 20th century saw the development of chemical compounds with improved safety profile. Conduction block with Ropivacaine in low doses displays greater sensory and motor separation, and a lower incidence of serious adverse effects make it the preferred drug in its class for peripheral nerve blockade . Adjuvants with ropivacaine are being used to improve the quality of block, to achieve a better intraoperative hemodynamics and to increase the duration of postoperative analgesia with minimal side effects . Adjuvants are pharmacological drugs that when co- administered with local anaesthetic agents may improve speed of onset as well as the quality and duration of analgesia. Various adjuvants have been used to improve the efficacy of nerve blocks including antagonist of NMDA receptor (ketamine, magnesium),GABA

agonist(midazolam),adrenergic agonist (clonidine, adrenaline), COX2 inhibitors(ketrolac) etc. Alpha 2 adrenergic agonists used as an adjuvants for peripheral nerve blockade produces better analgesic effects with minimum complication.Dexmedetomidine is a highly selective alpha2 adrenergic agonist with eight times greater affinity for alpha 2 than clonidine which is also an alpha adrenergic agonist. Analgesic requirements in post operative period greately reduced by the use of adjuvants .With the knowledge of pharmacological properties and drug interactions we designed a double blinded prospective randomized controlled study to study the effect of 0.75% ropivacaine alone and 0.75% ropivacaine with dexmedetomidine in axillary brachial plexus block using nerve locator .Our aim is to compare the onset and duration of sensory and motor block and side effects.

AIM AND OBJECTVES OF THE STUDY

The aim of the study is to compare the efficacy of 0.75% Ropivacaine along with combination of 0.75% ropivacaine and dexmedetomidine in axillary brachial plexus block for forearm and hand surgeries.

The objectives of the current study is to to compare the efficacy of 0.75% Ropivacaine along with combination of 0.75% ropivacaine and dexmedetomidine in axillary brachial plexus block with respect to

onset of sensory block

onset of motor block

duration of sensory block

duration of motor block

rescue analgesia

ANATOMY OF BRACHIAL PLEXUS

The brachial plexus supplies all of the motor and almost all of the sensory function of the upper extremity.

Brachial plexus Formation;

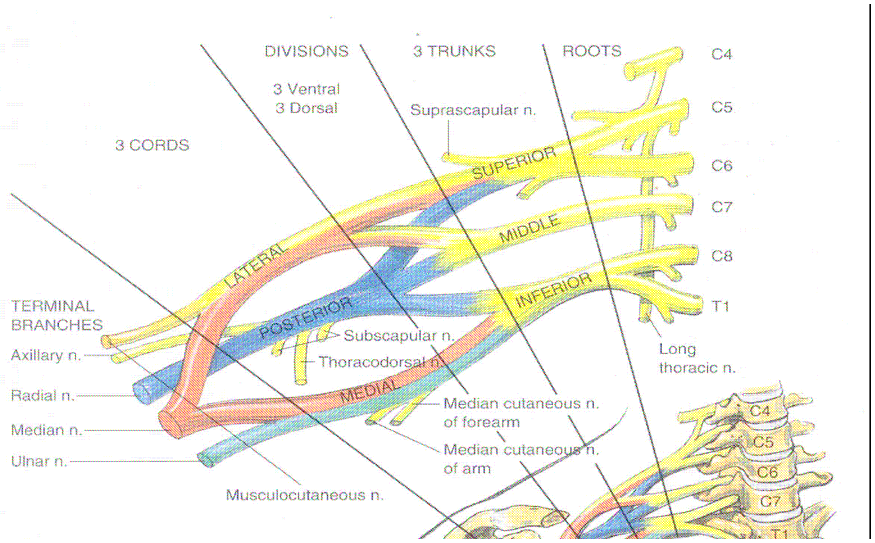


Figure –1 Brachial Plexus Formation

The plexus is formed from the anterior primary rami of fifth , sixth, seventh, eighth cervical and first thoracic nerve segments and frequently receives small contributing branches from the fourth cervical and second thoracic nerve .

Occasionally, the plexus receives contribution from C4, and the contribution from T1 is reduced or absent (pre-fixed plexus) .The post-

fixed plexus moves one segment lower than usual,here contribution from T2 is larger and from C5 is reduced or absent .

The roots arise from the intervertebral foramina. The five roots of the plexus after leaving the intervertebral foramina, travel laterally in the trough atop the transverse process of the cervical vertebra. They emerge between anterior scalene and middle scalene muscles and lie superior and posterior to the subclavian artery. Union of C5 and C6 roots forms the superior trunk. The middle trunk is formed by continuation of the C7 root. The inferior trunk is formed by the union of the C8 and T1 roots.The inferior trunk lies on the first rib posterior to subclavian artery, the middle and superior trunks are more superior .Each of the three trunks of brachial plexus divides into an anterior and posterior division. The six divisions moves into the axilla and there join up into three cords, lateral, medial and posterior, and are related to second part of the axillary artery.

Recombination of divisions form the cords they surround the axillary artery and named according to their position.

Cord formation :

1) The lateral cord gathers the anterior divisons of upper and middle trunks

2) The medial cord is the continuation of the anterior division of the lower trunk.

3) All three posterior divisions merge to form the posterior cord.

The cords descend into the axilla, where each has one major branch, in addition to several minor branches ,before becoming a major terminal nerve of the upper extremity. Branches of the lateral and medial cords form the median nerve. The lateral cord gives off a branch that forms the musculocutaneous nerve, whereas the posterior cord becomes the axillary and radial nerves.

The relations of the brachial plexus

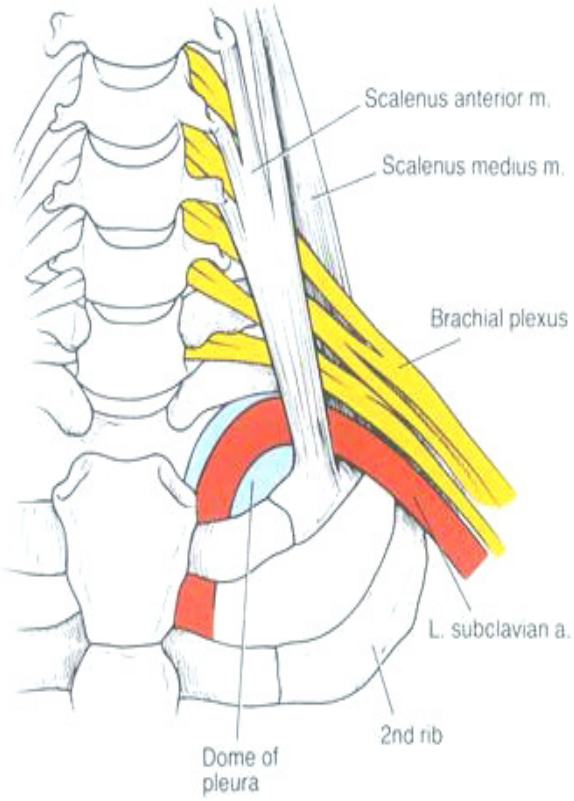


Fig-Relations of the Brachial plexus

ROOTS :

Roots of plexus emerge between the anterior an middle scalene muscle. The roots of the plexus lie above the second part of the subclavian .The prevertebral fascia continues laterally with the roots of the plexus and encloses the entire plexus, together with axillary artery in a fascial sleeve , the axillary sheath .

TRUNKS:

Trunks present in posterior triangle covered by prevertebral fascia lie superficially.

Trunks are crossed by inferior belly of omohyoid,external jugular nerves and the supraclavicular nerves.

DIVISION:

Each trunk slants obliquely downwards towards the middle third of the clavicle, it terminates into anterior and posterior divisons just behind clavicle.

CORDS:

All three cords of brachial plexus lies above and lateral to first part of the axillary artery. The medial cord crosses behind the artery to reach the medial side of the second part of the artery.

The posterior cord lies behind the second part of the artery and the lateral cord lies on the lateral side of the second part of the artery.Thus the cords of the plexus have the relationship to the second part of the axillary artey that is indicated by their names.

THE BRANCHES OF THE BRACHIAL PLEXUS

1) The roots give branches:

(a) Nerve to scalene and longus coli (C5–8);

(b) Branch to phrenic nerve (C5)

(c) Dorsal scapular nerve (C5);

(d) Long thoracic nerve of Bell (C5–7);

2) The **trunks** give:

(a) Nerve to subclavius (C5, 6);

(b) Suprascapular nerve (C5, 6).

3) The **cords** give:

(a) Lateral cord

Lateral pectoral nerve (C5–7);

Musculocutaneous nerve (C5–7);

Lateral head of median nerve (C6, 7)

(b) Medial cord

Medial pectoral nerve (C8, T1);

Medial cutaneous nerve of arm (C8, T1);

Medial cutaneous nerve of forearm (C8, T1);

Medial head of median nerve (C8, T1);

Ulnar nerve (C7–8, T1);

(c) Posterior cord

Upper subscapular nerve (C5, 6);

Nerve to latissimus dorsi (C6–8);

Lower subscapular nerve (C5, 6);

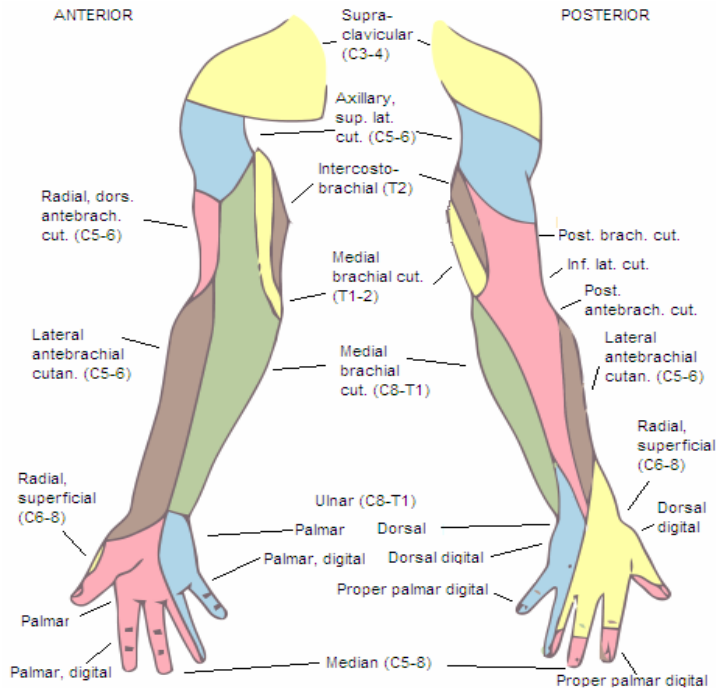
Axillary nerve (C5, 6);

Radial nerve (C5–8, T1).

In the axilla, median nerve lies lateral to the axillary artery,radial nerve posterior and ulnar nerve lies medial.

The axillary and musculocutaneous nerves exit the axillary sheath high up in the axilla, the musculocutaneous nerve travelling through the substance of the corachobrachialis muscle before becoming subcutaneous below the elbow.

SENSORY INNERVATION OF UPPER LIMB



AXILLARY BLOCK LANDMARKS :

1) The chief landmark is the axillary artery.The terminal portion is identified by the pulsations in the apex of axilla.The axillary artery at this point runs parallel to the axis of the humerus .
2) Insertions of tendons of pectoralis major and corachobrachialis form a second landmark.A line connecting these two points lies just below the point where the cords of the brachial plexus give origin to the nerves of arm.
3) The humerus forms the third landmark and is the backstop against which the other more pliable structures can be moved or steadied.

TECHNIQUE OF AXILLARY BLOCK

1) Nerve stimulation technique;

Identify the axillary artery with the arm abducted to 90 □ and the elbow flexed. Draw a line down from the anterior axillary fold (insertion of pectoralis major) crossing the artery .Fix the artery between index and middle finger.22 guage Insulated needle of length 3.5-5cm connected to the nerve locator ,after aseptic precautions,a skin wheal is raised over proximal portion of the artery,the needle is inserted in a two step ,four injection process with puncture at locations just superior and inferior to the axillary artery.With nerve stimulation technique,the nerves serving the area of proposed surgery are sought first. The median and musculocutaneous nerves lie on the superior aspect of the artery. ulnar nerve below the artery and radial nerve below and behind the artery. The stimulating needle introduced just below the axillary artery to elicit motor response from the radial nerve(thumb abduction) and the motor response of ulnar nerve (thumb adduction,little finger flexion)and 10-15 ml of local anaesthetic injected after negative aspiration .The needle is resited and advanced superficial to axillary artery for a median nerve motor response (index/middle finger flexion)and 5-8ml of local anaesthetic deposited after negative aspiration. The needle is taken out up to the level

of skin in the same insertion site redirect needle towards corachobrachialis to obtain musculocutaneous nerve motor response (elbow flexion) and inject about 5-8 ml of local anaesthetic solution. Stimulation of the target nerve at a current of 0.5 mA or less suggests accurate needle placement for local anaesthetic injection.

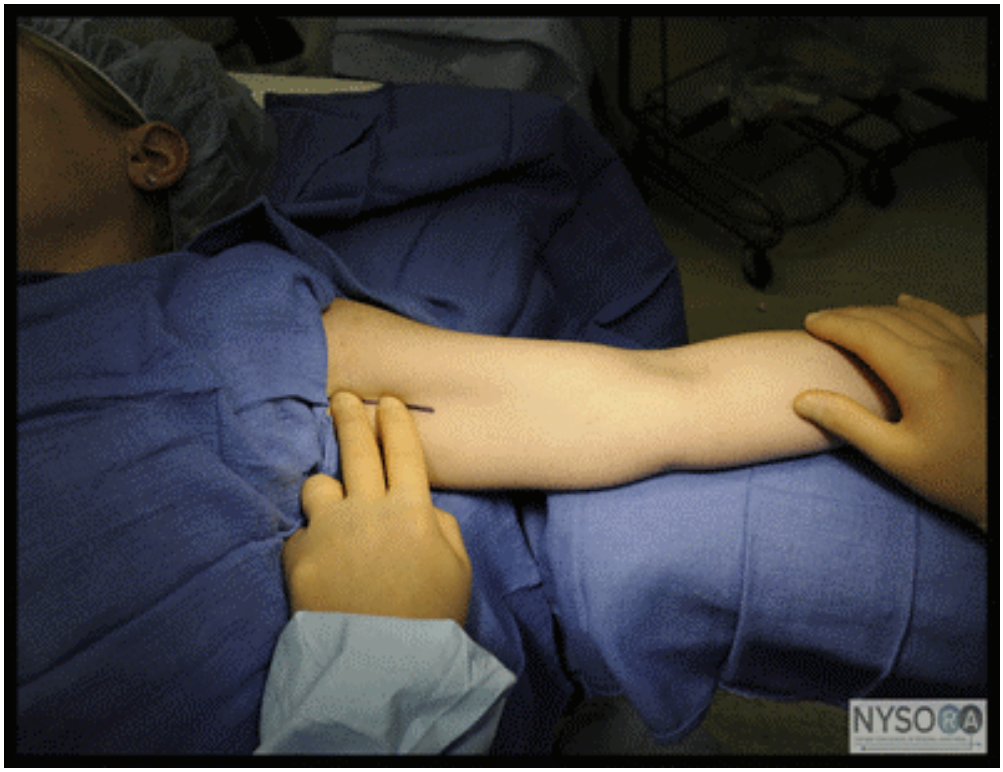


Fig-3Fixation of Axillary Artery

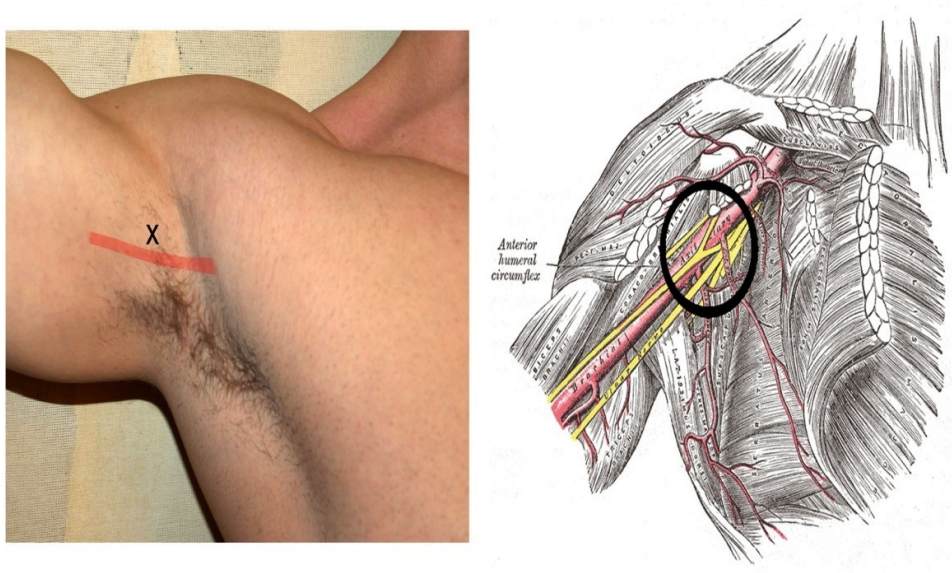


Fig-4 axillary artery surface marking

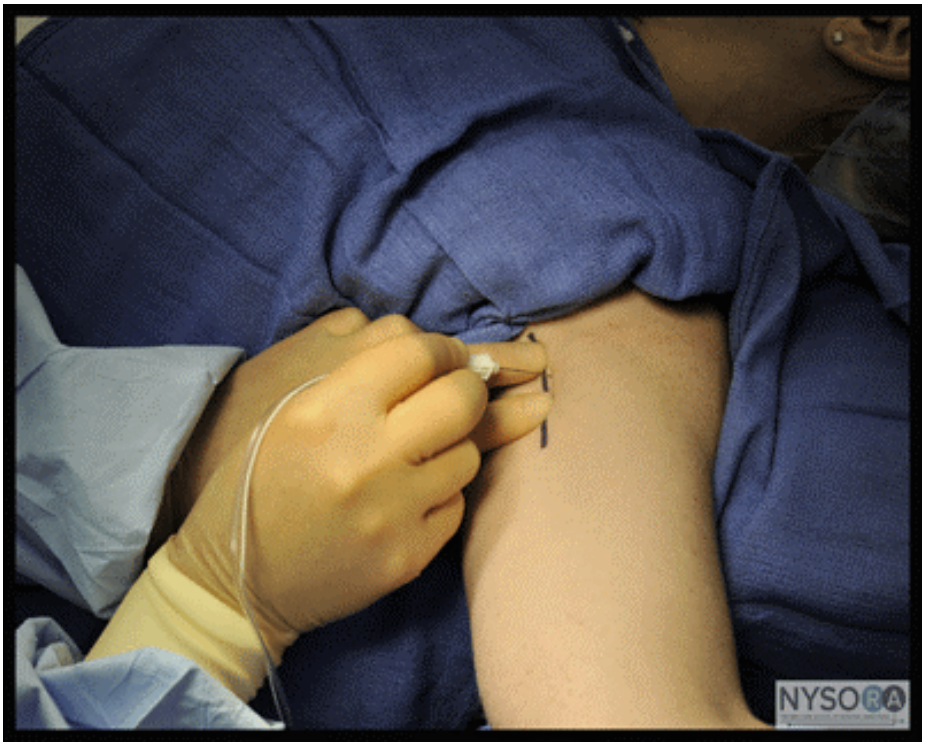


Fig-5 Axillary Block using nerve stimulator

ALTERNATIVE TECHNIQUES :

Paraesthesia Elicitation

Transarterial approach .

Loss of resistance –click/pop on entering fascial sheath.

Field block of the brachial plexus with fan like injection

Ultrasound guidance

PERIPHERAL NERVE

The peripheral contains both afferent and efferent fibres, which are bundled in to one or more fascicles and organized within three tissue layers .

Endoneurium – loose connective tissue surrounds individual nerve fibres within each fascicle .

Perineurium –collagenous connective tissue surrounds each fascicle .

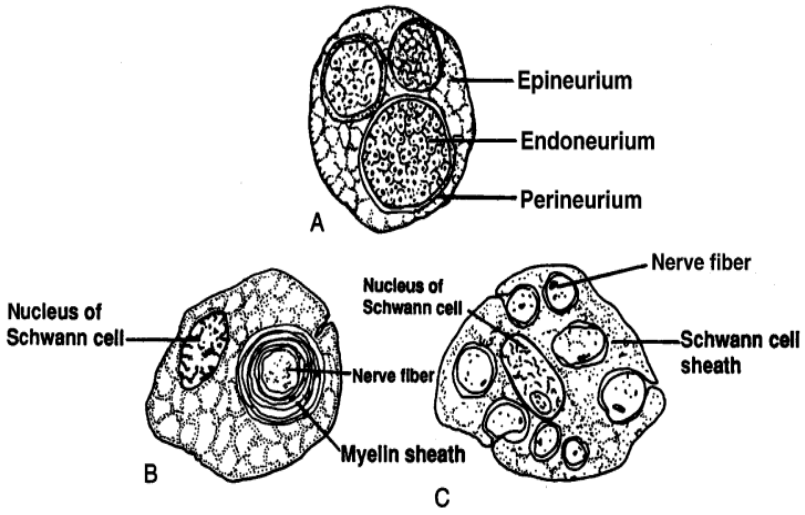
Epineurium-dense connective tissue encases groups of fascicles into a cylindrical Sheath.

These layers provide protection to the surrounded nerve fibers and act as barriers to passive diffusion of local anaesthetics.Nerve axons were classified as myelinated and unmyelinated.

Nerve axons are myelinated if individual axons are surrounded by Schwann cells interrupted at nodes (nodes of Ranvier, which are axonal areas free of myelin at fixed intervals) and unmyelinated if several axons are surrounded by a Schwann cell forming a Remak bundle.

Non myelinated nerves contain many axons encased in single Schwann cell sheath,in contrast all motor and sensory fibres are enclosed in many layers of myelin,which contains plasma membranes of specialized Schwann cells that wrap themselves around the axon.

Myelin sheath interrupted at short regular intervals by specialized regions called nodes of Ranvier. The Na+ channels that serve generation and propagation of impulses are highly concentrated at the nodes of Ranvier of myelinated fibres but are distributed all along the axon of nonmyelinated fibers. Action potentials pass thorough myelinated axons by saltatory conduction.



CLASSIFICATION OF NERVE FIBERS

Fibre type (Erlanger & Gassers)	Fibre diameter (µm)	Conduction velocity (m/s)	Spike duration (ms)	Absolute refractory period (ms)	Function
A					
alpha	12-20	70-120	0.4-0.5	0.4-1	Proprioception, somatic motor
beta	5-12	30-70			Touch, pressure, motor
gamma	3-6	15-30			Motor to muscle spindles
delta	2-5	12-30			Pain, cold, touch
B	<3	3-15	1.2	1.2	Preganglionic autonomic
C					
Dorsal root	0.4-1.2	0.5-2	2	2	Pain, temperature, Mechanoreception, Reflex response
Sympathetic	0.3-1.3	0.7-2.3	2	2	Postganglionic sympathetic

Tab-1

Greater is the diameter of a given nerve fiber, greater is the speed of conduction .

Relative susceptibility of mammalian nerve fibers to conduction block produced by local anaesthetics - C > B > A

PHYSIOLOGY OF NERVE CONDUTION

Nerve impulse is a transient wave of electrical excitation that travels from point to point down the length of nerve fibre.The interior of cells is electronegative and exterior as electro positive.

This electrochemical difference is caused by difference in ionic composition between cytoplasm and extracellular fluid. Intracellular concentration of potassium is about 150 mEq/L(range-110-170 mEq/L) with a sodium concentration of only 5 mEq/L(range 4-10mEq/L). Extracellular fluid concentration of these ions are reversed.

A special protein in the membrane Na+/K+ pump actively transports K+ into the cell and Na+ out of cell.A selective permeability of the resting nerve membrane to potassium ions but is relatively impermeable to sodium, this help to maintain a negative electrical potential of approximately -90 mv.

Action potential initiated by local membrane depolarization. During the passage of impulse membrane momentarily loses its selective resistance to electrochemical pressure of the sodium ions on the outside , thus sodium enters the axon and causes membrane potential to fall to a

critical threshold level of – 50 to -60 mv, whereupon the membrane becomes completely depolarized and permeable to sodium ions.

An explosive influx of sodium thus occurs at this point and electrical current generated then spread the loss of selectivity to the next section of membrane and the impulses travel down the length of fiber by self depolarization and regeneration.

In non myelinated fibers the impulse is propagated in a continuous depolaroization of the adjacent membranes.In myelinated fibers the impulse propagation is discontinuous and progresses by jumping from node to node.

The nature of conduction is summarized as a depolarization-repolarization process.

Flow of ions responsible for action potential is mediated mainly by voltage gated sodium channels.In absence of stimulus voltage gated sodium channels exist in resting or closed state.

On membrane depolarization conformational change in this channel converting it in to open state. Within milliseconds after opening channels undergo a transition to inactivated state.

Depending on the frequency and the voltage of the initial depolarizing stimulus, the channel may undergo either fast or slow inactivation. Fast inactivation completes with in a millisecond and is sensitive to action of local anaesthetics. Slow inactivation lasting for

seconds to minutes and resistant to actions of local anaesthetics. Nine isoforms of voltage gated sodium channels have been identified.

MOLECULAR MECHANISM OF LOCAL ANAESTHETIC

Local anaesthetics block impulses by interfering with the function of sodium channels. Although several local anaesthetics can bind to other receptors like voltage gated potassium channels and nicotinic acetyl choline receptors, and their amphipathic nature may enable them to interact with plasma membranes it is widely accepted that they induce anaesthesia and analgesia through interaction with the sodium channels.

TONIC BLOCKADE :

Application of local anaesthetics typically produce a concentration dependent decrease in the peak sodium current known as tonic blockade. It reflects the reduction in the number of sodium channels for a given drug concentration present in the open state in equilibrium.

USE DEPENDENT BLOCKADE :

Repetitive stimulation of the sodium channels often leads to a shift in steady state of equilibrium, resulting in greater number of channels being blocked at the same drug concentration. Mechanism for this blockades not completely understood, Two theories put forward 1) Modulated receptor theory –local anaesthetics bind to open or

inactivated channels more avidly than the resting channels suggesting that drug affinity is a function of a channels conformational state.

(2) Guarded receptor theory –intrinsic binding activity remains essentially constant regardless of a channels conformation.

CARVINO describes the mechanism how the local anaesthetics inhibit the conduction of impulses across the nerves, local anaesthetics exists in both charged and uncharged forms. The relative concentration of these two forms depend on the pKa of the solution, pH of the site where it is injected. Charged form is active form and responsible for local anaesthetic action, uncharged forms responsible for diffusion across liphophillic membranes across the cell. Drugs from inside of cells act on the sodium channels. They occupy specific receptors on the innerside of sodium channel and inhibits the conduction of ions through them ,thus the cell remains in the state of persistent depolarization. This inhibits the propagation of action potential.

Channel narrowing and membrane expansion are other probable site of actions due to nonspecific absorption across the cell membrane .

The surface charge theory :

This theory states that penetration of axonal membrane by lipophillic portion of the local anaesthetic drug and neutralization of

axolemmal negative charges on surface are by the positively charged terminal amino group of drug. Acquired positive charges counteract the electronegativity of the external membrane.This results in increase in the transmembrane potential without altering much of the intracellular resting potential. This inhibits the conduction of nerve impulses from the normal areas to anaesthetized areas of the nerve membrane. Thus it produces a conduction block across two portions. According to surface charge theory the active form of local anaesthetic drug is the charged form of drug.

THE LOCAL ANAESTHETIC PROCESS SEQUENCE :

Onset And Establishment Of Anesthetic Block

1) Diffusion –movement of molecules of local anaesthetic agents

   To nerve cell compartment

   To vascular compartments

   Depends on water solubility and dissociation to free undissociated base.

2) Penetration-entrance through cell membrane

   Depends on lipophillic polar group

3) Distribution-Movement into nerve bundle core and mantle

   Depends on concentration gradient from extracellular to intracellular space.

4) Fixation-Physiochemical combination of local agent with lipoproteins in membrane.

   Depends on molecular configuration and ionization

   Lipophillic pole is oriented in the cell membrane

   Hydrophillic pole is oriented toward axoplasm

REVERSAL AND RECOVERY

5) Absorption-Entrance into vascular compartment

   Initially excess anaesthetic in extracellular space enters capillaries

   Depends on concentration gradient between extracellular space and vascular compartment

6) Reversal-as equilibrium is established, the concentration in the centre of nerve bundle becomes highest and gradient then changes direction to the extracellular, interstitial and vascular compartments.

7) Redistribution-Drug in plasma is distributed to regions beyond the site of injection.

8) Destruction-Hydrolysis occurs in plasma, conjugation in liver And Elimination- Elimination of free and conjugate products in kidney.

FATE OF LOCAL ANAESTHETIC AGENTS :

Drug - Local disposition --> systemic absorption --> systemic disposition

1) Bulk flow of solution	vascular distribution
2) Diffusion of molecule	extravascular distribution
3) Binding	excretion
4) Metabolism	metabolism
Local effects	systemic effect
(therapeutic)	(side effect)

MINIMUM ANAESTHETIC CONCENTRATION :

Lowest concentration of an anaesthetic agent that blocks the conduction of nerve impulses in a reasonable time is termed as Cm or minimum anaesthetic concentration. Each local anaesthetic has a specific cm for a given diameter nerve fiber.

DIFFERENTIAL BLOCKADE :

On basis of differing cm values of local anaesthetics for different nerve fibers, selective blockade of certain fibres and their function without blockade of other fibers can be accomplished. This is called differential blockade.

In peripheral nerves the small pain fibers of the A delta fibers and the C fibers are readily blocked and at lower concentration than the A motor, proprioceptive, and sensory fibers .

TRANSITIONAL BLOCK :

A latent period is required for a local anaesthetic to change the function of a nerve from an unblocked state to the blocked state. During this time one conceives of either a partial or threshold block and repetitive stimuli may be conducted. This state is called Wedensky block.

CLINICAL ORDER OF NERVE BLOCK :

Vasomotor

Cold

Warmth

Slow pain

Fast pain

Motor

Joint sense

Pressure

The order of return of function is essentially the sequence of return of motor activity, sensory activity, and finally sympathetic vasomotor activity.

NERVE STIMULATOR GUIDED PERIPHERAL NERVE BLOCK

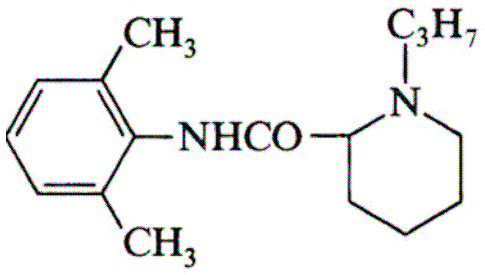
Peripheral nerve stimulation technique was originally described in 1911. The availability of high quality insulated needles and stimulators designed for peripheral nerves provide a practical adjuvant to plexus and peripheral nerve blocks in the arm and elsewhere in the body. Peripheral nerve stimulator allows localization of peripheral nerves without the need for elicitation of a paraesthesia. Anode terminal of the stimulator connected to the patient and cathode terminal connected to the stimulating needle. The syringe with local anaesthetic connected to the flexible tubing attached with the needle. A skin wheal with local anaesthetic created at the point of entry of the needle, intial current set at 2 mA with frequency of 2 hertz after eliciting target contraction or the desired response with 0.5 mA. Once the needle has been successfully located which is determined by persistence of motor twitch with 0.5 MA, local anaesthetic is injected after careful aspiration in incremental volumes of 5 mls. A threshold current of 0.5 mA is usually associated with successful block.Existence of motor twich with 0.2 mA current and resistance while injecting drug signifies intraneural placement of the needle which warrants the withdrawl of needle.

PHARMACOLOGY OF ROPIVACAINE

Ropivacaine is a long acting amide local anesthetic similar to bupivacaine in structure and pharmacodynamics. It is formulated as a single levorotatory enantiomer rather than a racemic mixture.

Chemical Name: S-(-)-1-propyl-2', 6'-pipecoloxylidide hydrochloride monohydrate.

STRUCTURAL FORMULA



Molecular formula : C17H26N2O•HCl•H2O

Molecular weight : 328.89.

MECHANISM OF ACTION

Ropivacaine reversibly blocks the entry of sodium into the nerve cell membranes, leading to decreased membrane permeability to sodium and raises the threshold for nerve excitability, thereby slowing the nerve conduction and reduces the rate of rise of the action potential.


7.5mg/ml
ROPIN 0.75%
0.75%

PHARMACODYNAMICS

Ropivacaine has fewer propensities for cardiac and CNS adverse effects because of its stereo selective property. It has similar efficacy of levo bupivacaine and bupivacaine in blocking peripheral nerve. When given neuraxially (epidural or intrathecal) it is less potent than bupivacaine. It is associated with incidence of lower grade motor blockade when compared to bupivacaine. Because of its lower grade motor blockade, it has reduced potential for CNS and cardiac adverse effects. It is currently a new agent of choice for regional anaesthesia.

PHARMACOKINETICS

The plasma concentration varies depends on the dose, route of administration and injection site vascularity. Ropivacaine follows linear pharmacokinetics Cmax is proportional to the dose. When given extradural its absorption is biphasic (t1/2 is 4mins and 4 hrs) and complete. Elimination of ropivacaine is mainly depends on absorption which is a rate limiting step. Epidurally given drug has longer t1/2 compared to intravenous route. The terminal half life of ropivacaine is1.8hrs after IV route. Ropivacaine is highly protein bound particularly to alpha1-acid glycoprotein and only 6% present as unbound fraction. It

crosses easily through the placenta and degree of binding to the plasma proteins in fetus is less compared to mother.

METABOLISM

It is metabolized mainly by aromatic hydroxylation particularly in liver. If given IV, about 86% of the doses are excreted via urine out of which only 1% is unchanged fragment. The main metabolite is 3-hydroyropivacaine excreted after conjugation. Other metabolites are 4-hydroxyropivacaine and 2', 6'-pipecoloxylidide. The PPX(2', 6'-pipecoloxylidide) has longer t1/2 and lower clearance after infusion through epidural.

Clearance – unbound ropivacaine – 13.94L/h/Kg

Clearance – Total ropivacaine – 0.555L/h/Kg

Volume of distribution – 65.57L/min

Terminal t1/2 of ropivacaine - 3.3hrs

Terminal t1/2 of PPX – 17.8 hrs

INDICATIONS

Local infiltration

Epidural anesthesia and analgesia

Spinal anesthesia

Peripheral nerve blocks

CONTRAINDICATIONS

Hypersensitivity

Intravenous regional anaesthesia

Paracervical block in obstetric anaesthesia

Hypovolemia

Premature children

DOSAGE AND ADMINISTRATION

Caudal – 1mg/kg, 0.2% or 2mg/ml produces a level belowT12.

Epidural block with 6-14 ml of 0.2% ropivacaine provide adequate analgesia.

Spinal – 2-3ml of 0.75 %(7.5mg/ml) with doses between 15-22.5mg results in sensory block upto T4 or T5

peripheral nerve blocks-3mg/kg.

ADVERSE EFFECTS:

Hypersensitivity reactions

Main effects - Hypotension

bradycardia

nausea, vomiting

fever

paresthesia, headache

pruritus , rigors

back pain

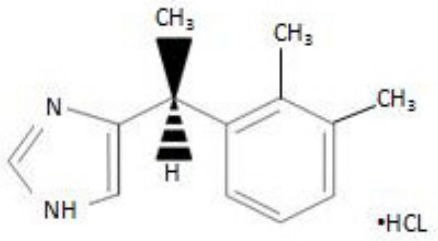
Less common side effects : CNS toxicity, cardiac toxicity.

PHARMACOLOGY OF DEXMEDETOMIDINE

Dexmedetomidine is an alpha-2-adrenoceptor agonist with sedative, anxiolytic, and analgesic properties.The U.S. Food and Drug Administration (FDA) first approved dexemedetomidine for use in adults in 1999. Now it is widely used as sedative, adjunct analgesia for various diagnostic procedures.

Physio Chemical Charecteristics :

Dexmedetomidine hydrochloride is the d-enantiomer of medetomidine chemically described as (+)-4-(d)-[1-(2,3-dimethylphenyl) ethyl]-1H-imidazole monohydrochloride, an imidazole derivative. Its molecular weight is 236.7,empirical formula is C13H16



Dexmedetomidine is a highly selective alpha-2-adrenergic receptor agonist.

Adrenergic receptors mediate the effects of catecholamine and are classified as alpha or beta adrenoceptors.Alpha receptors are located pre-,

post-, and even extrasynaptically. They are subclassified as alpha-1 and alpha-2 adrenoceptors, based on the binding affinity of the antagonists yohimbine and prazosin. Three alpha-2-adrenoceptor agonist subtypes, alpha-2A, alpha-2B, and alpha-2C, have been identified by radioligand binding studies and subsequently confirmed by genetic cloning. Alpha-2 adrenoceptors are cell membrane receptors composed of a single polypeptide chain. They are part of the G protein-coupled receptor superfamily.The three subtypes are only 50% conserved at the amino acid level, but exhibit 72-75% identity in their membrane-spanning hydrophobic domains, arranged as alpha helices, and are the site for ligand binding.The particular arrangement of amino acids within these segments determines ligand specificity.

MECANISM OF ACTION :

1) Activate inhibitory action of G proteins leading to decrease in cyclic AMP.
2) Activate G proteins which directly act on membrane bound ion channels, particularly potassium channels.
3) Activate nitric oxide, cyclic GMP pathway by inhibiting the release of noradrenaline within neuronal tissue.

In the dorsal motor complex of medulla it causes hypotension and bradycardia.

Action on locus coeruleus leads to sedation and analgesia .

Alpha 2 receptors are G protein–coupled receptors in the transmembrane region of the central and peripheral nervous system particularly at autonomic ganglion of presynaptic and postsynaptic region. Nor epinephrine and clonidine acts on these receptors inhibit the enzymes adenyl cyclase and phospholipase C resulting in the inhibition of calcium ion entry and facilitation of opening potassium ion channels outwards that results in hyperpolarization.

Presynaptic activation of α 2 adrenergic receptors in sympathetic nerve endings and nor adrenergic neurons inhibit norepinephrine release.

In central nervous system it leads to inhibition of sympathetic activity, resulting in hypotension,bradycardia,and sedation.But at higher doses it causes hypertension through receptor activation present in ssmooth muscle cells of the resistance vessels.Hypnosis through α2 receptors of locus coeruleus,analgesia mediated by α 2 receptors in spinal cord.Imidazoline ring of α2 agonists like clonidine and dexmedetomidine interact with imidazoline receptors.

PHYSIOLOGICAL FUNCTIONS OF ALPHA 2 RECEPTORS;

α2a - presynaptic feedback inhibition of norepinephrine release Hypotension, Sedation ,analgesia, inhibition of epileptic seizures.

α2b – hypertension, analgesic effect of nitrous oxide placental angiogenesis.

α2c - feedback inhibition of adrenal catecholamine release modulation of behaviour.

Pharmacokinetics of dexmedetomidine :

Administration

The most common route of dexmedetomidine administration is intravenous, other routes are possible and include intranasal, buccal, peroral, transdermal, and intramuscular administration.The level of bioavailability is highest for the buccal route (82%), followed by the intramuscular and transdermal routes (73% and 51%, respectively).

Distribution

Dexmedetomidine has a rapid distribution phase, with a distribution half-life of about six minutes.The steady-state volume of distribution is approximately 97 ± 29 liters. It binds to serum albumin and alpha- glycoprotein, with an average protein binding of 94%. There is no

sex difference in terms of protein-binding ability. The fraction of dexmedetomidine that is bound to plasma proteins is significantly lower in subjects with hepatic impairment compared to healthy subjects. There is no reported significant drug-drug interaction due to changes in protein-bounding ability, and dexmedetomidine has been explored in vitro in combination with different common highly protein-bound drugs including fentanyl, ketorolac, theophylline, lidocaine, and warfarin.

Metabolism

Dexmedetomidine undergoes almost complete biotransformation by the liver with very little unchanged dexmedetomidine excreted in the urine and feces. Hepatic biotransformation involves both direct glucuronidation and cytochrome P450 mediated metabolism. The hepatic extraction ratio of dexmedetomidine has been estimated to be about 70%. The major metabolic pathways of dexmedetomidine include

1) direct N-glucuronidation to inactive metabolites;
2) aliphatic hydroxylation (mediated primarily by CYP2A6) of dexmedetomidine to generate 3-hydroxy-dexmedetomidine, the glucuronide of 3-hydroxy-dexmedetomidine, and 3-carboxy dexmedetomidine;

3) N methylation of dexmedetomidine to generate 3-hydroxy N-methyl-dexmedetomidine, 3-carboxy N-methyl-dexmedetomidine, and dexmedetomidine-N-methyl O-glucuronide.

Dexmedetomidine has been shown to have a mild inhibitory effect on CYP2D6-dependent dextromthorphan O-demethylase activity. Therefore, noclinically significant drug interaction was demonstrated with other drugs that are metabolized by CP450 enzymes.

Elimination:

Terminal elimination half-life (t½) of dexmedetomidine is approximately two hours, and clearance is estimated to be approximately 39 ± 10L/hr.Ninety-five percent of the drug is present as metabolites (34% products of N-glucuronidation; 14% products of aliphatic hydroxylation, including 3-hydroxy dexmedetomidine, the glucuronide of 3-hydroxy-dexmedetomidine, and 3-carboxylic acid-dexmedetomidine; 18% products of N-methylation, including 3 hydroxy N-methyl dexmedetomidine, 3-carboxy N-methyl dexmedetomidine, and N methyl O glucuronide dexmedetomidine; 28% unidentified) in the urine and 4% in the feces.

No unchanged dexmedetomidine is detected in the urine. Approximately 85% of the metabolites recovered in the urine are excreted within 24 hours after the infusion.

Pediatric patients :

The pharmacokinetics of dexmedetomidine in pediatric patients is similar to those found for adults.Volume of distribution (Vd) 1.5-2.2l/kg, clearance rate (Cl) 0.56-1l/kg/hr, and terminal t½ 1.6-2.7 hours. However, the CL in neonates was just 18.19 l/hr/70 kg. This was attributed to immature enzymatic pathways. The values approach those reported for adults by one year of age.

Age and Gender :

Dexmedetomidine doesnot show any variation in pharmacokinetics in both the sexes and in all age groups.

Hepatic impairment :

Patients with severe hepatic failure showed a significantly increased Volume of distribution,and decreased terminal half-life and clearance values of dexmedetomidine compared with age-matched controls; therefore, the pharmacokinetics of dexmedetomidine were markedly affected by hepatic insufficiency.So it is necessary to reduce the dose in patients with hepatic derangements.

Renal Impairment :

Pharmacokinetics of dexmedetomidine in subjects with normal renal function, and those with mild, moderate and severe renal impairment, as defined by creatinine clearance, did not differ, as it affects only the excretion of metabolites.

Drug Interactions :

No evidence of cytochrome P450 mediated drug interactions that are likely to be of clinical relevance.

Pregnancy, Labour and Lactation :

There were no adequate and well controlled trails, hence should be used with caution.

Pharmacodynamics of dexmedetomidine :

1) Cardiovascular system :

Dexmedetomidine can produce either hypo- or hypertension. At lower doses, their dominant action is sympatholysis , which is mediated by the alpha-2A-adrenergic receptor subtype.There are several well-documented mechanisms for this activity, including the inhibition of the firing of the locus coeruleus (the pivotal noradrenergic replay nucleus in the brain stem) and inhibition of nerepinephrine release at the neuro-effector junction.Central and peripheral sympatholytic effects of alpha-2 adrenoceptor stimulation may be augmented further by the inhibition of ganglionic transmission. At higher doses, the hypertensive action dominates via the activation of alpha-2B adrenoceptors, which are located on the smooth muscle cells in the resistance vessels.

2) Respiratory system :

Dexmedetomidine causes a slight increase in PaCO2 . The increase in PaCO2 is no different from that during normal sleep. In addition, dexmedetomidine does not produce significant respiratory depression or decrease arterial oxygen saturation.

Nevertheless, dexmedetomidine is able to induce some aspects of natural sleep.

The results are similar for pediatric patients. Several prospective studies have found minimal or no change in respiratory function during the administration of dexmedetomidine .

3) Central nervous system

a) Sedation

The locus coeruleus, the predominant noradrenergic nucleus in the brainstem, provokes the sedative and hypnotic response to alpha-2 agonists by reducing the firing of neurons. Dexmedetomidine induces sedation that is qualitatively different from that produced by other commonly used sedative agents. Despite being deeply sedated, while subjects were able to perform a complex task without loss of accuracy, provided a rousing stimulus was applied.

Dexmedetomidine produces profound anxiolysis in surgical patients. In some studies, the anxiolytic effect was comparable with that obtained with benzodiazepine premedication.

4) Analgesia :

The primary site of analgesic action is thought to be spinal cord. Dexmeditomidine also inhibits the release of substance P from the dorsal horn of the spinal cord,leading to primary analgesic effect.

5) Gastric emptying :

Dexmedetomidine delays gastric emptying and gastrointestinal transit, but the degree of this effect is trivial when compared with the effect that morphine has on these two functions.

6) Salivation and secretion levels are also reduced

7) Eyes

The level of intraocular pressure is reduced and mydriasis develops.

ADVERSE EFFECTS :

Most frequently observed sideeffects are

hypo or hypertension

dry mouth, nausea

dizziness, headache

bradycardia , arrhythmias

AV block, extra systoles

pulmonary oedema.

ALPHA 2 ANTAGONIST :

Atipamezole, a selective α2 adrenoceptor antagonist readily reverses the sedative properties of dexmedetomidine. Intravenous atipamezole reverses the sedation and sympatholysis in dose dependent manner.

REVIEW OF LITERATURE

1) **Feroz Ahmad Dar et al(2013);** studied the effects of addition of alpha 2 adrenergic agonist Dexmedetomidine to ropivacaine for axillary brachial plexus blockade.They divided 80 patients into groups ,group R(40 ml of 0.5% ropivacaine+1 ml of normal saline),group RD(40 ml of 0.5% ropivacaine with 50µg of dexmedetomidine).They found that sensory and motor block onset times were shorter in group RD than group R .Similarly the duration of sensory and motor block were also longer in RD than R. The blood pressure and heart rate also lower in RD group than those R group.They noticed bradycardia in 7 patients in RD group while none in R group. They concluded that addition of dexmedetomidine as adjuvant with Ropivacaine for axillary block decreases the onset time as well as duration of sensory and motor block.

2) **Marhofer et al**; (2012) (Dexmedetomidine as an adjuvant to ropivacaine prolongs peripheral nerve Block):

This study evaluates the effect of adding a alpha 2 adrenergic receptors agonist Dexmedetomidine to ropivacaine for peripheral nerve block.36 patients undergo ultra sonagram guided ulnar nerve block divided into three groups. Group R who received 3 ml ropivacaine 0.75% and those who received 3 ml of 0.75%+ 20µg of dexmedetomidine and group RSD who received 3 ml of ropivacaine 0.75%+20µg IV. They found that no difference in sensory onset time between the groups, while the motor onset was significantly faster in dexmedetomidine groups. Sensory and motor duration was found to be similar. They concluded that the addition of dexmedetomidine increase the duration of sensory and motor block and improve the block quality.

3) **F.W.Abdalla et al**; (Facilitatory effects of perineural dexmedetomidine on neuraxial and peripheral nerve block) : 2013.

This meta-analytic study evaluate the addition of dexmedetomidine along with local anaesthesia for perineural nerve block and neuraxial block.They analysed all randamoized control study analyzing the effect of dexmedetomidine as an adjuvant for peripheral and neuraxial blocks. They found that sensory block duration was prolonged by 150 min with neuraxial dexmedetomidine. Duration of sensory block with perineural dexmedetomidine found to be prolonged (284 minutes), though this finding was statistically insignificant.Time for rescue analgesia and motor block duration were found to be prolonged. Bradycardia was noted in 7% patients.They concluded that the addition of alpha 2 receptor agonist dexmetomidine as an adjuvant to local anaesthetic had a facilitatory effect on the sensory onset time and motor duration when given in brachial plexus block.

4) **Yu Zhang et al**; (2014) (Perineural administration of dexmedetomidine in combination with ropivacaine prolongs axillary brachial plexus block)

This double blinded prospective control study evaluates the hypothesis of dexmedetomidine to ropivacaine prolongs axillary brachial plexus block. Forty-five patients of ASA I~II and aged 25-60 yr who were scheduled for elective forearm and hand surgery were randomly divided into 3 equal groups and received 40 ml of 0.33% ropivacaine + 1 ml dexmedetomidine (50 μg) (Group DR1), 40 ml of 0.33% ropivacaine + 1 ml dexmedetomidine (100 μg) (group DR2) or 40 ml of 0.33% ropivacaine + 1 ml saline (group R) in a double-blind fashion. The onset and duration of sensory and motor blocks and side effects were recorded. The demographic data and surgical characteristics were similar in each group. Sensory and motor block onset times were the same in the three groups. Sensory and motor blockade durations were longer in group DR2 than in group R ($P < 0.05$). There was no significant difference in the sensory blockade duration between group DR1 and group R. Bradycardia, hypertension and hypotension were not observed in group R and occurred more often in group DR2 than in group DR1. They concluded that

Dexmedetomidine added to ropivacaine for an axillary brachial plexus block prolongs the duration of the block.

5) **Sarita S Swami et al (2012)** (Comparison of dexmedetomidine and clonidine (alpha 2 agonist drugs) as adjuvant to local anaesthesia in supraclavicular brachial plexus block):

This randomized double blinded prospective control study compares the addition of clonidine and dexmedtomidine to local anaesthetic as a adjuvant for supraclavicular block.60 patients divided into two groups. Group C (clonidine1µg/kg) and group D (dexmedetomidine 1µg/kg)

Added to bupivacaine 0.25%.Duration of sensory and motor block in group D is greater than group C(413.97 in group C vs 472.24 minutes).Onset time of sensory and motor block between groups were found to be statistically insignificant.The duration of analgesia was found to be twice more with group D than group C(456±97 minutes vs 289±62 minutes) respectively.They found that the quality of block was also improved with group D.They concluded that addition of dexmedetomidine as an adjuvant to supraclavicular block improves the quality of block and prolongs the duration and analgesia when compared to clonidine.

6) **Kenan Kaygusuz et al;** (2012) (Effects of Adding Dexmedetomidine to Levobupivacaine in Axillary Brachial Plexus Block)

This randomized double blinded prospective control studyevaluates the effect of dexmedetomidine with levobupivacaine in axillary block. The patient receiving levobupivacaine and isotonic saline categorized as group L and dexmedetomidine with levobupivacaine group categorized as group D.Results analysia shows shorter onset time with group D when compared to group L.The rescue analgesic time,duration ofsensory and motor duration were longer in group D when compared to group L.Haemodynamic parameters found to be stable.No adverse effects seen.They concluded that the addition of alpha 2 agonist like dexmedetomidine to axillary block improves the block quality,shortens the sensory onset time and increased the sensory and motor duration as well as analgesia.

7) **Yu-Nan Lin et al, (2013) (**Addition of dexmedetomidine to ropivacaine improves cervical plexus Block) (2013)

Conducted a randamoised double blinded prospective control study. They studied 40 (ASA) Class I or IIadult patients who were scheduled to undergo thyroid surgery were randomly allocated to the following groups to receive cervical plexus block: 30 mL of 0.375% ropivacaine combined with 1 mg kg_1 of dexmedetomidine; 30 mL of 0.375% ropivacaine combined with saline (control). The sensory block onset time, duration of analgesia, mean arterial pressure (MAP), heart rate (HR), and the incidences of side effects, such as hypotension, bradycardia, and hypoxemia were recorded.By the end of study the results showed that addition of dexmedetomidine to ropivacaine (Group D) shortened the sensory block onset time compared with the ropivacaine group (Group C) (95% confidence interval [CI] 4.18e5.26; $p < 0.05$). The duration of analgesia of cervical plexus block in Group D was significantly longer than that in Group C . MAP level and HR level in Group D were significantly lower than that in Group C ($p < 0.05$).So they conclude that addition of 1 mg kg_1 dexmedetomidine to ropivacaine for cervical plexus block could shorten the sensory block onset time and extend the duration of analgesia, and increased the quality of analgesia."

8) **Harshavardhana et al, (2014)** Efficacy of Dexmedetomidine Compared to Clonidine added to Ropivacaine in Supraclavicular Nerve Blocks(2014))

This study evaluates that dexmedetomidine along with 0.5% ropivacaine produces an effective analgesia and motor block when compared clonidine.100 patients grouped in to two groups 50 each,group RD and group RC.Group RD receives specific alpha 2 agonist dexmedetomidine and ropivacaine whereas group RC receives ropivacaine with clonidine. The results showed that RD group had an increased motor and sensory duration when compared to RC.They concluded that dexmedetomidine is an better adjuvant to ropivacaine than the Clonidine.

9) **Malenfant Rancourt et al, (2012)** (Posterior tibial nerve sensory blockade duration prolonged by adding dexmedetomidine to ropivacaine).(2012)

This prospective, randamoised,controlled double blinded crossover trail analysed the effect of addition of dexmedetomidine with ropivacaine for tibial nerve block.14 healthy volunteers selected divided into two groups.group R received 0.5% ropivacaine 10 ml,group RD receives 10ml of a solution containing 0.5% ropivacaine with 1 μg/kg of dexmedetomidine.crossover study done.The primary end point was the duration of sensory blockade. Secondary outcomes were the onset time and the adverse effects such as hypotension,bradycardia,hypoxia and sedation. They found that sensory block lasted longer in group RD than in group R(21.5 VS 16.2 hrs) $p<0.001$. Onset time similar between two groups. Mean systolic and diastolic blood pressure was stable through out the study period in R group but decreased in RD group.Heart rate similar in both groups. So they concluded that dexmeditomidine added to ropivacaine for tibial block prolongs the duration of sensory block with similar onset time.

10) **Obayah et al (2010)** Addition of dexmedetomidine to bupivacaine for greater palatine nerve block prolongs postoperative analgesia after cleft palate repair)

In this study children underwent cleft palate repair had given greater palatine block for postoperative pain relief.The study evaluates wheter the addition of alpha 2 adrenergic agonist like dexmedetomidine with bupivacaine improves the quality and postoperative analgesia.The children divided in to two groups, group B receives bupivacaine alone, while group BD receives bupivacaine along with dexmedetomidine. The duration of postoperative analgesia and time for rescue analgesia were found to be increased with dexmedetomidine adjuvant group than the bupivacaine alone group.

MATERIALS ANDS METHODS

The study was conducted at chengalpattu medical college between 2013-2014. After obtaining ethical committee approval,60 ASA I- II patients posted for elective upperlimb surgeries are randomly allotted into two groups

Study design : A Prospective randomized double-blinded study.

Sample size : 60 patients were selected and allocated in two groups randomly.

Inclusion Criteria :

ASA PS I & II

Age 18 to 60 for upper limb forearm and hand surgeries

Both sexes

Exclusion Criteria:

Patient Refusal

Patient with history of bleeding disorders

Patients on anticoagulation therapy

Patients with documented neuromuscular disorders

Patient with known allergy to local anaesthetic drugs

Psychiatric illness

Preop preparation :

Patients who satisfy the inclusion criteria were selected Informed consent obtained from all the patients. Preoperative evaluation including detailed history, clinical evaluation, investigations and airway assessment were done Visual Analogue Scale (VAS) was explained in detail to the patients in the preoperative period.

Premedication :

All patients premedicated with inj glycopyrolate 0.2 mg iv 40min before surgery. Patients also received midazolam o.1 mg/kg before the procedure.

Materials used :

Nerve stimulator with 22G 50mm long short bevel, free tip, insulated needle

10 ml syringe

20 ml ampoule of 0.75% ropivacaine

1 ml ampoule of 100 mic dexmedetomidine

Monitoring :

Continuous ECG, intermittent non invasive blood pressure monitoring , Spo2 monitoring done.

METHODOLOGY

After preop evaluation, written informed consent,premedications patient shifted inside the theatre. intravenous access was done using 18 G venflon and ringer lactate infusion was started.preoperative heart rate, Spo2,blood pressure was obtained.

Patients assigned into two groups :

Group R-Recieves 25 ml volume of 0.75% ropivacaine and 1 ml normal saline.

Group D-Recieves 25 ml volume of 0.75% ropivacaine and 1ml(50 microgram) of dexmedetomidine .

The anaesthetist who prepared the drug combination did not participate in the monitoring or assessment of the patient.The person who performed the axillary block as well as monitoring was blinded to the groups the patients belongs.

Patients in both groups are placed in supine position with upperlimb to be blocked kept with the arm abducted to 90 □ and the elbow flexed.

Group R received axillary brachial plexus block using nerve stimulator guidance. After obtaining a motor response of ulnar, radial and

median nerve with output current of 0.8-0.4mA, using negative aspiration 25 ml of 0.75% ropivacaine was delivered . Immediately after injection has been stopped; the arm was kept adducted and the hand resting on the chest.

Patients in group D received axillary brachial plexus block using nerve stimulator guidance.After obtaining motor response of ulnar, radial and median nerve with output current of 0.8-0.4mA, using negative aspiration a drug combination of 25 ml of 0.75% ropivacaine and 50microgram of dexmedetomidine was delivered Immediately after injection has been stopped; the arm was kept adducted and the hand resting on the chest.

The following parameters were observed following the block.

1) Hemodynamic parameters :

Pulse rate, non invasive blood pressure, oxygen saturation are monitored. Mean arterial blood pressure (MAP) and pulse rate (PR), oxygen saturation will be recorded before application of the block as well as immediately after block & 5 min intervals until 30 min & with 30 min intervals thereafter, until the end of the operation. Any drop in blood pressure morethan 20% from the baseline signifies hypotension and

managed with inj ephedrine 6 mg.Any decrease in pulse rate of less than 60 beats /min managed with inj.atropine 0.004mg/kg I.V

2) Sensory block :

Sensory block tested with a 22-gauge hypodermic needle by using the pinprick test and compared with the same stimulation in the contra lateral hand. Sensory block tested every 1 min.

Time to Onset of Sensry Block (minute)is defined as the time between the end of last injection and the total abolition of the pinprick response, and complete paralysis in all sensation over hand and forearm

3) Motor block :

Motor block accessed according to modified bromage scale for upper extremity 0-able to raise the extended arm to 90 □ for full 2 seconds; 1-able to flex the elbow and move the fingers but unable to rise the extended arm ; 2-unable to flex the elbow but able to move the fingers , 3- unable to move arm,elbow or fingers .Assessed at 1 minute interval until complete motor blockade occurred.

Onset of motor blockade is defined as the time taken from the injection of drug to development of complete motor block (bromage score 3)

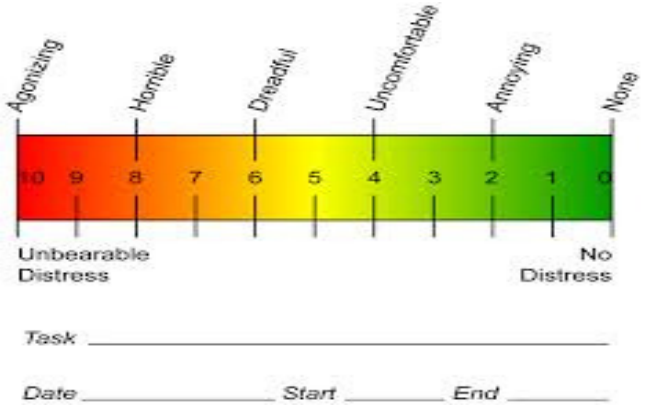
Duration of sensorial block (minute): Time interval between withdrawal of the needle and reappearance of paresthesia in the 4 nerve distribution areas.

Duration of motor block (minute): defined as the time interval between the onset of motor block till the complete regression of motor block.

First analgesic requirement time (minute): Rescue analgesia is defined as the time interval between block placement and patient's first analgesic request.

Postoperatively pain scores were recorded by using visual analogue score between 0 to 10(0-no pain,10= most severe pain)

Rescue analgesia given at VAS score of 4 or above



During the intraop period and postoperative period patients monitored for side effects like hypotension, bradycardia, hypoxia, nausea, vomiting .

OBSERVATION AND RESULTS

The following observations were made and datas were collected. Pulse rate, blood pressure, Spo2 at 5 min intervals until 30 min & with 30 min intervals thereafter, until the end of the operation thereafter at 1 hr intervals.

Onset time of sensory block

Onset time of motor block

Duration of sensory blockade

Duration of motor blockade

Duration of analgesia

Untoward side effects

STATISTICAL ANALYSIS

Data were analysed using SPSS16.0V,software.two sided independent students t tests to analyse continuous data and chi square test for categorical data were used. $P<0.05$ was considered as statistically significant.

DEMOGRAPHIC DATA

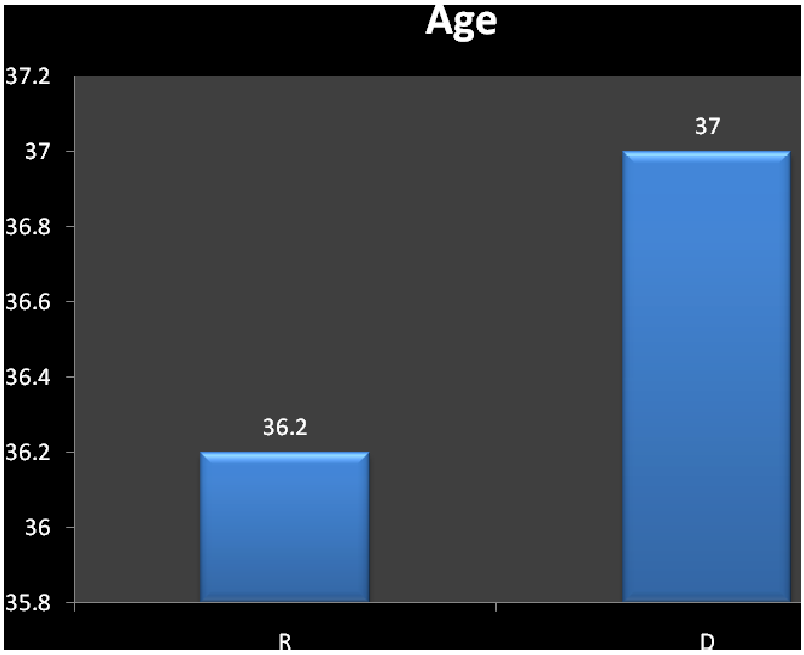
The two groups were comparable with respect to their age, weight, sex, and ASA physical status. There is no statistically significant difference among two groups in demographic profile.

AGE (student's t test)

	No of cases	**Mean ± S.D (minutes)**	**P value**
Group R	30	36.2±12.881	0.390 Not significant
Group D	30	37±12.268	

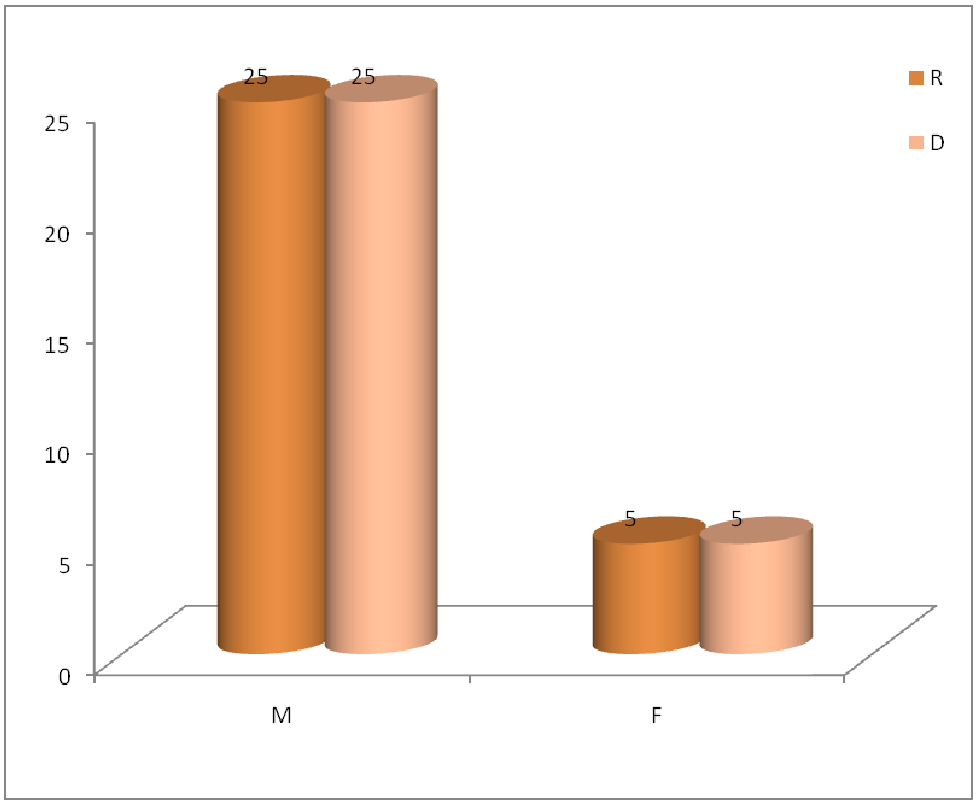
The mean age in years was 36.2±12.881(minutes) in group R and 37±12.268(MINUTES) in group D.There was statistically no significant difference between two groups (p>0.05) .

AGE GROUPS



SEX DISTRIBUTION

	Male	**Female**	**Total**
Group R	25	5	30
Group D	25	5	30
TOTAL	50	10	60

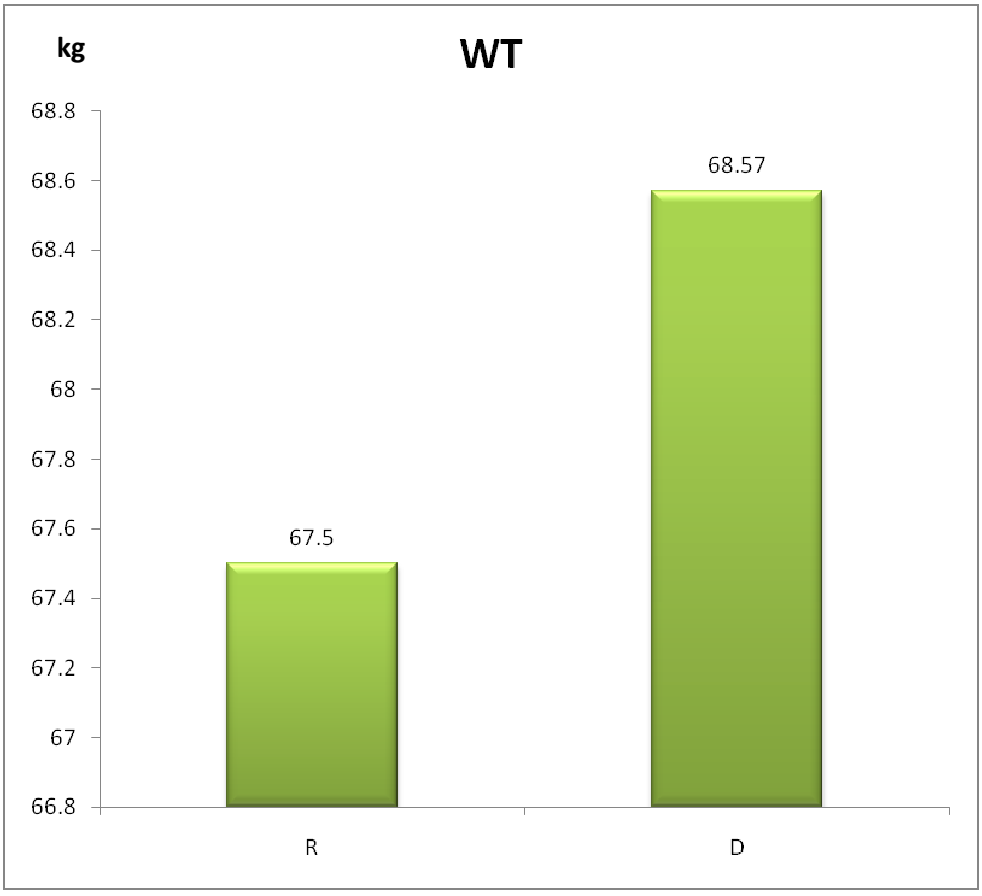


SEX

WEIGHT (Student's t test)

	No of cases	**Mean ± S.D (Kilograms)**	**P value**
Group R	30	67.5 ± 2.596	0.08
Group D	30	68.57±1.9950	

The mean weight in kilograms was 67.5±2.596(Kg) in group R and 68.57± 1.9950(Kg) in group D.Both groups did not differ significantly.

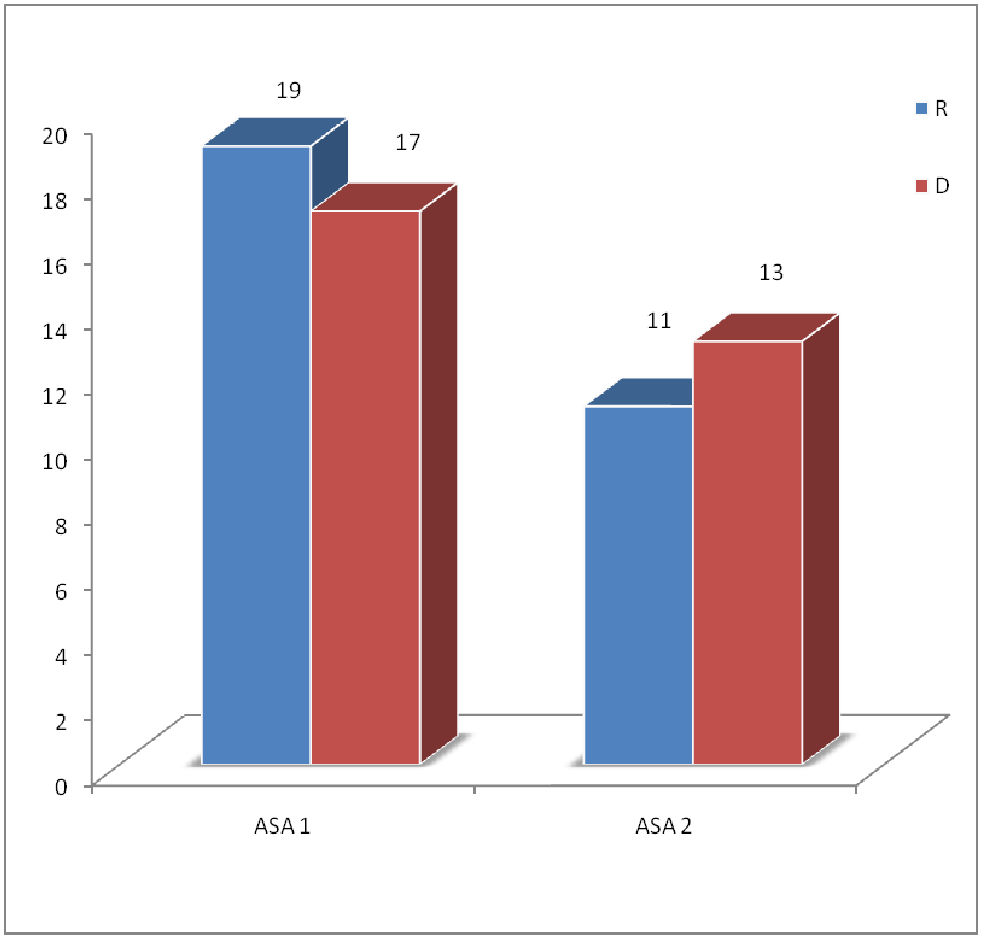


GROUPS

ASA PHYSICAL STATUS

	ASA –PS I	ASA –PS II	P value
Group R	19	11	0.596
Group D	17	13	

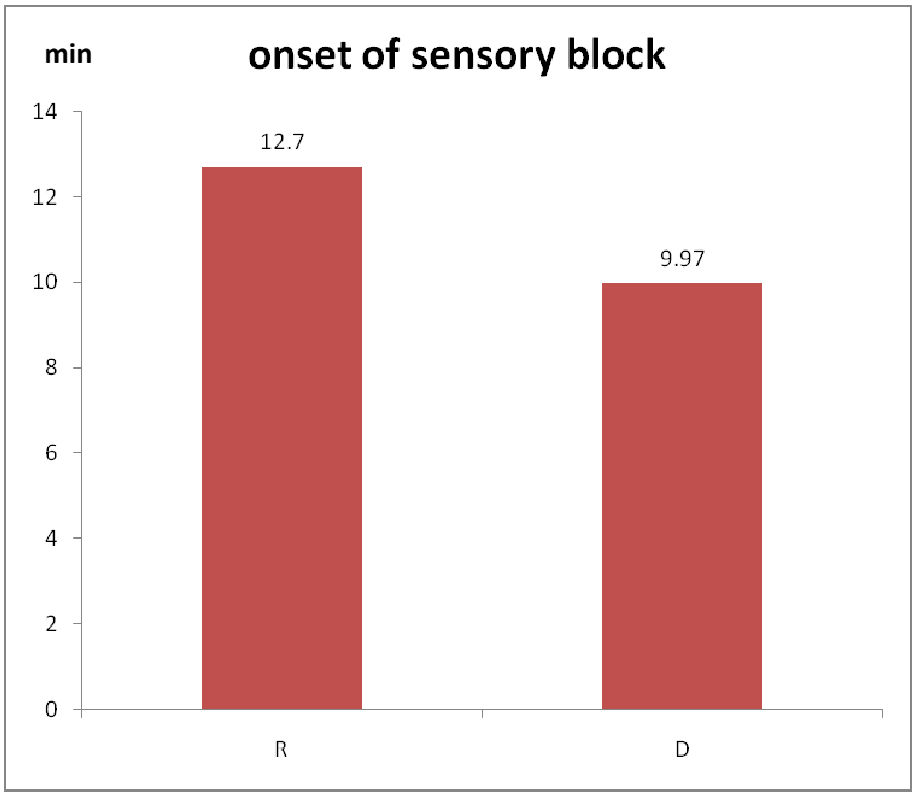
There is no significant difference between both the groups


19
17
11
13
20
18
16
14
12
10
8
6
4
2
0
R
D
ASA 1
ASA 2

ONSET OF SENSORY BLOCK

	Mean (min)	S.D	P value
Group R	12.7	1.343	0.001
Group D	9.97	0.928	

The mean time for onset of sensory block is 12.7±1.343 (minutes) in group R and 9.97±0.928(minutes) in group D.There was significant difference among two groups in the time for onset of sensory block.($p<0.05$)

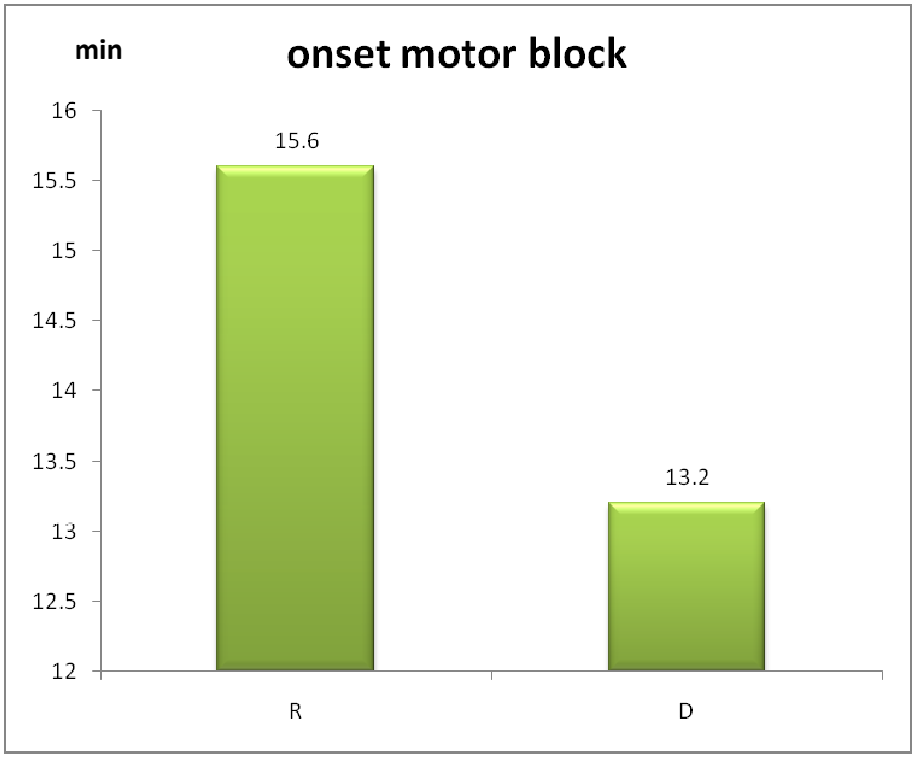


GROUPS

ONSET OF MOTOR BLOCK

	Mean(min)	S.D	P value
Group R	15.6	1.589	0.034
Group D	13.2	0.925	

The mean time for onset of motor block is 15.6±1.589(min) in group R and 13.2±0.925(min) in group D.There was significant difference among two groups in the time for onset of motor block. ($p<0.05$)

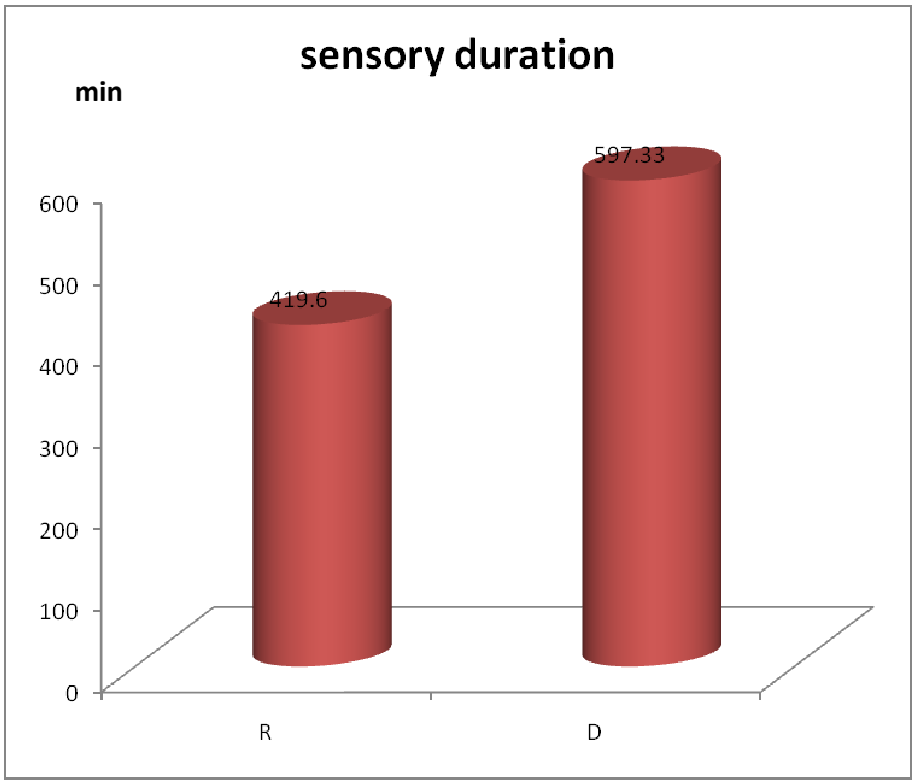


GROUPS

SENSORY BLOCK DURATION

	Mean(min)	S.D	P value
Group R	419.6	9.665	0.0028
Group D	597.33	10.807	

The mean duration of sensory block is 419.6±9.665 (minutes) in group R and597.33±10.807 (minutes)in group D.There was significant difference among two groups in the sensory block duration .($p<0.05$)

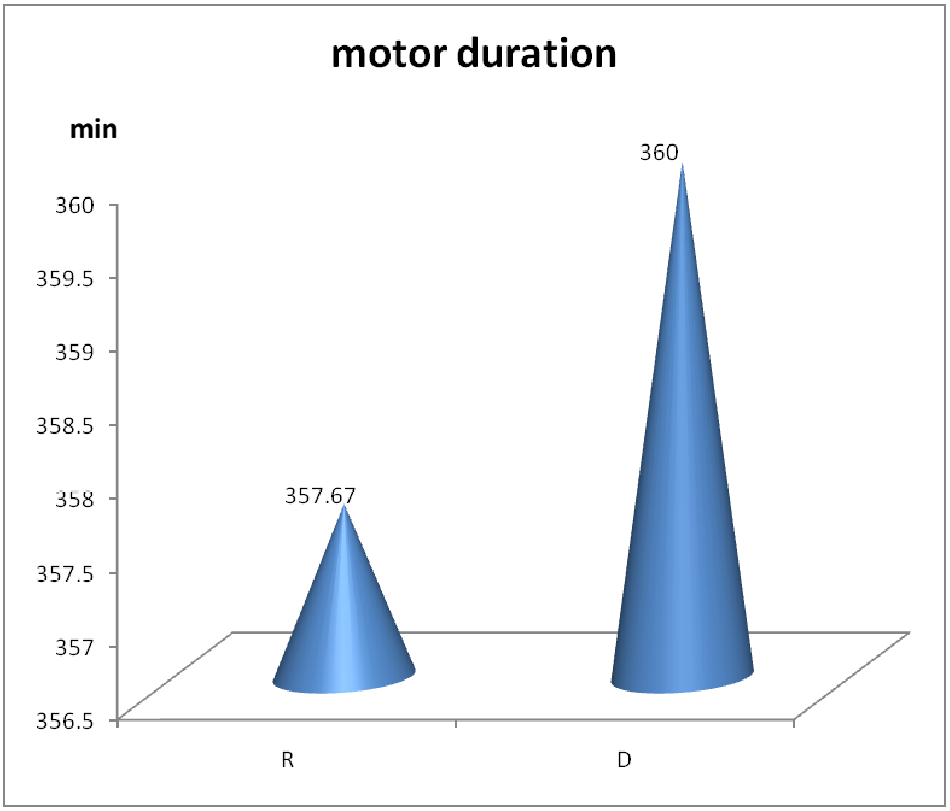


GROUPS

MOTOR DURATION

	Mean(min)	S.D	P value
Group R	357.67	9.71	0.32
Group D	360	8.3	IN SIGNIFICANT

The mean duration of motor block is 357.67±9.71 (minutes)in group R an360.00±8.3(minutes) in group D.There was no significant difference among two groups in the motor block duration ($p>0.05$).

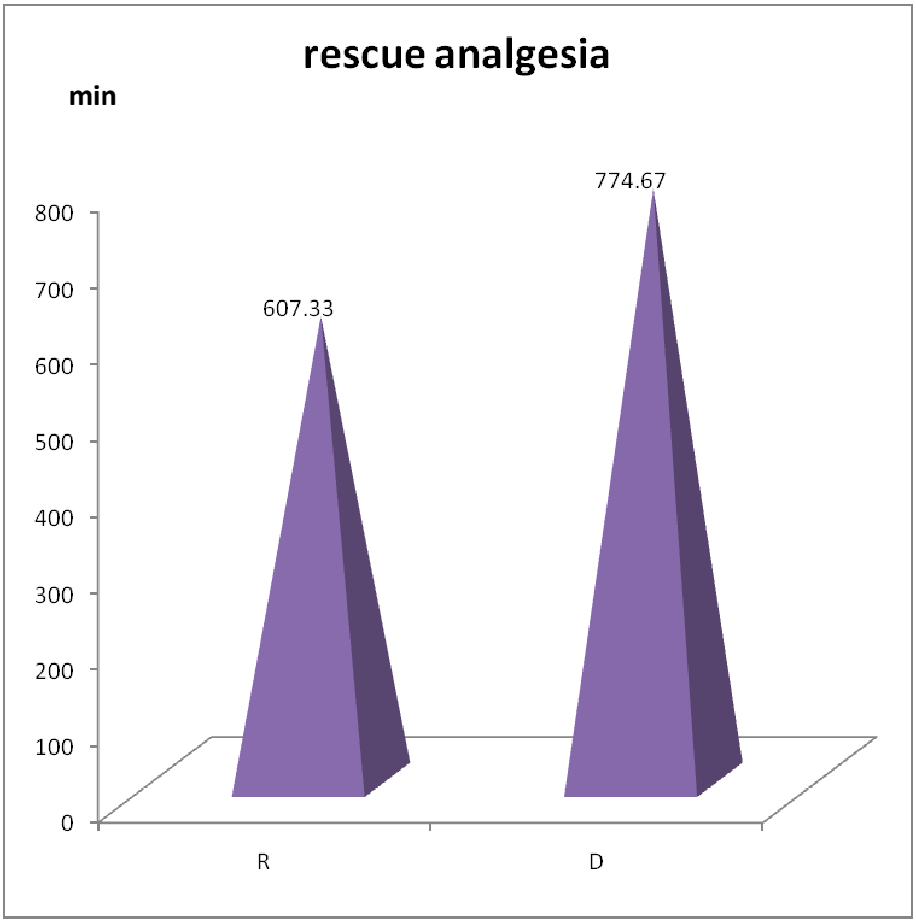

motor duration
min
360
359.5
359
358.5
358
357.5
357
356.5
357.67
360
R
D

GROUPS

RESCUE ANALGESIA

	Mean(min)	S.D	P value
Group R	607.33	13.629	0.001
Group D	774.67	10.743	

The mean time for the need of rescue analgesia is 607.33 ± 13.629 (min) in group R and 774.67 ± 10.743 (min) in group D. There was significant difference among two groups in the mean time for the need of rescue analgesia .($p<0.05$)


rescue analgesia
min
800
700
600
500
400
300
200
100
0
607.33
774.67
R
D

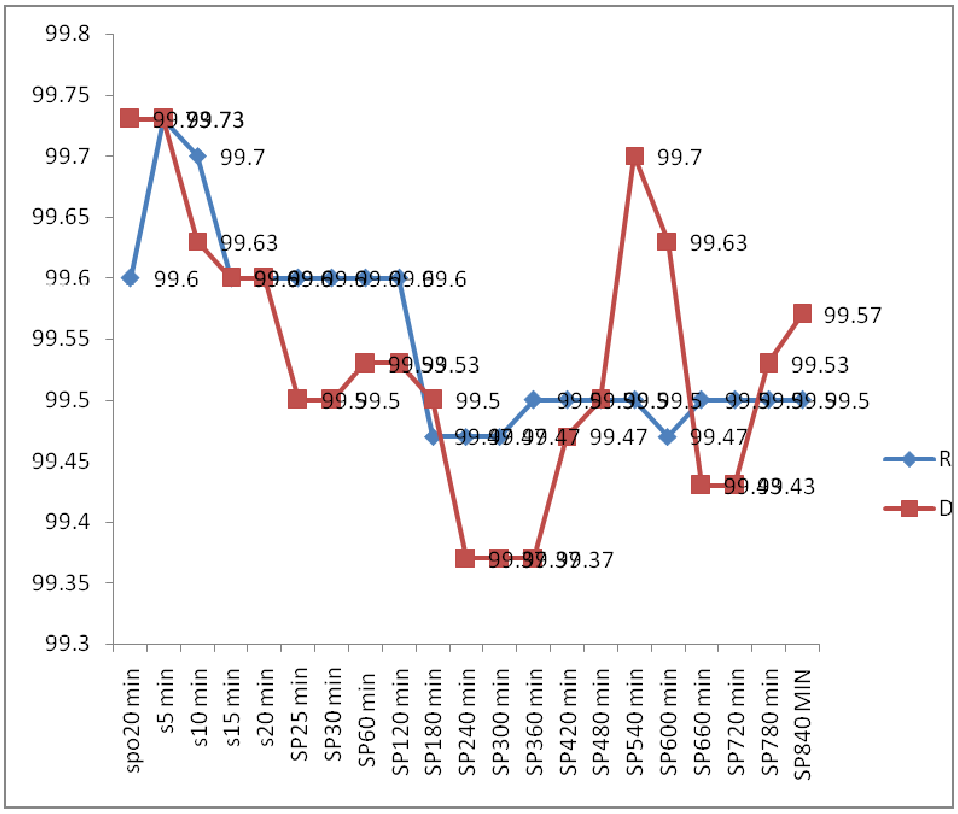
GROUPS

O2 SATURATION

Intraoperative	No of cases	Mean ±S.D(%)	P value
Group R	30	99±0.0046	0.9602
Group D	30	99±0.0026	
Postoperative	No of cases	Mean±S.D	
Group R	30	100±0.001	
Group D	30	99±0.00111	

There was no statistical significance between two groups in saturation both during intraoperative and postoperative period.(P>0.05)

O2 SATURATION

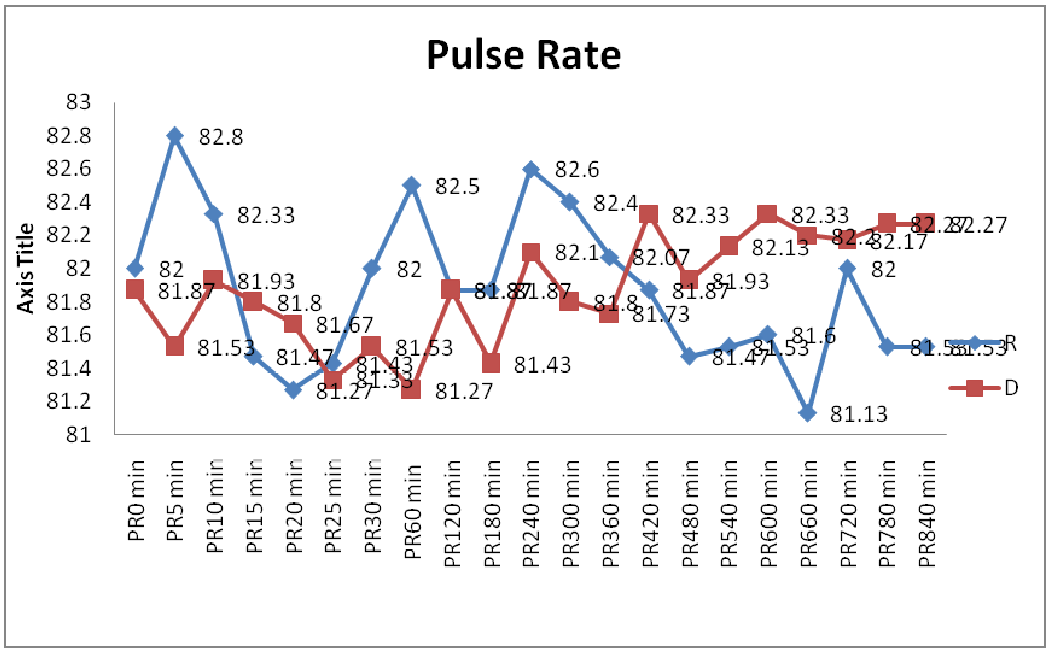


TIME

PULSE RATE

	Mean(min)	S.D	P value
Group R	81.87	1.737	0.343
Group D	81.43	1.775	

The mean pulse rate in group R was 81.87±1.737 and in group D was 81.43±1.775,which was not found to be statistically significant (P>0.05).

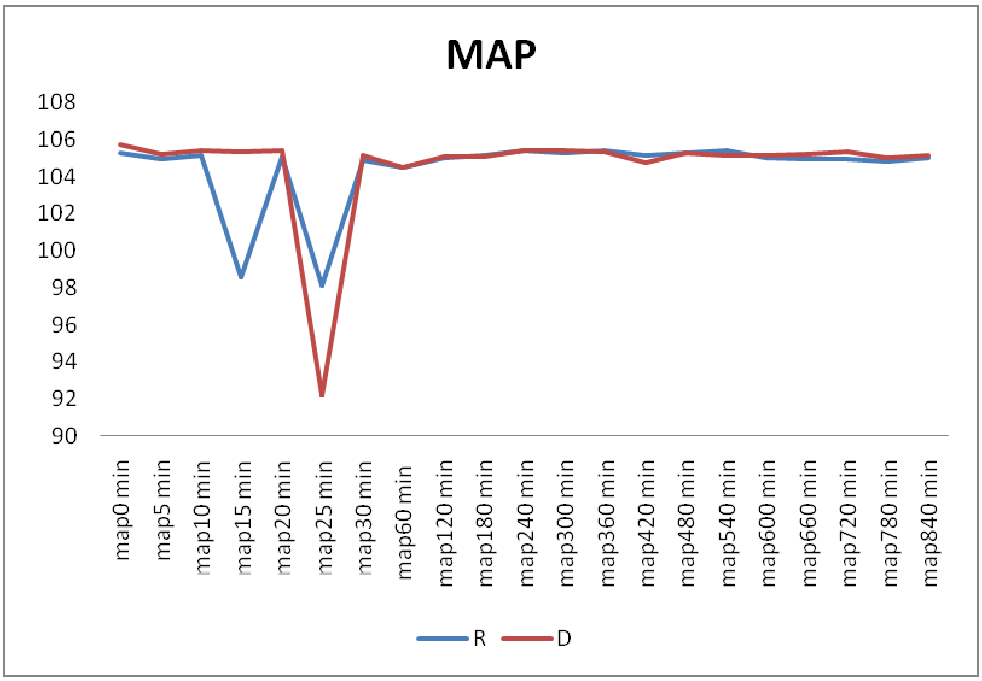

Pulse Rate
Axis Title
83
82.8
82.6
82.4
82.2
82
81.8
81.6
81.4
81.2
81
PR0 min
PR5 min
PR10 min
PR15 min
PR20 min
PR25 min
PR30 min
PR60 min
PR120 min
PR180 min
PR240 min
PR300 min
PR360 min
PR420 min
PR480 min
PR540 min
PR600 min
PR660 min
PR720 min
PR780 min
PR840 min
R
D

MEAN ARTERIAL BLOOD PRESSURE

	Mean(mm/Hg)	S.D	P value
Group R	105.3	2.748	0.962
Group D	105.07	2.703	

The mean arterial pressure in group R was 105.03±2.748 (mmHg) and in group D was 105.07±2.703(mm/Hg) which was not statistically found to be significant.

MAP


MAP
108
106
104
102
100
98
96
94
92
90
map0 min
map5 min
map10 min
map15 min
map20 min
map25 min
map30 min
map60 min
map120 min
map180 min
map240 min
map300 min
map360 min
map420 min
map480 min
map540 min
map600 min
map660 min
map720 min
map780 min
map840 min
R
D

TIME

DISCUSSION

Peripheral nerve blocks provide excellent anaesthesia,it markedly decreases the postoperative analgesic requirement.Adjuvants are used along with local anaesthesia to improve the quality of block .An ideal adjuvant should provide a longer duration of analgesia and better hemodynamic stability. Alpha 2 agonists show promising results with local anaesthetics in increasing the block duration and quality. Abdallah et al showed that perineural dexmedetomidine as an local anaesthetic adjuvants can prolong the duration of analgesia when compared to local anaesthesia alone. Animal studies showed that perineural dexmedetomidine added to bupivacaine or ropivacaine prolong the duration of sensory and motor block various studies evaluated the effects of dexmedetomidine on various nerve blocks and reported to be safe and effective. In our study 50 µg(1 ml) of dexmedtomidine was added to 25 ml of 0.75% ropivacaine or 25ml of 0.75% ropivacaine +1 ml of normal saline added. The efficacy of dexmedetomidine as an adjuvant in axillary brachialplexus block was studied in 30 patients in each group who underwent elective upperlimb forearm and hand surgeries.

The patients in both groups with respect to age, weight, ASA Physical status did not show Statistically significant difference.

Onset of sensory block :

The study had shown that addition of 50 µg of dexmedetomidine to 25 ml of 0.75% ropivacaine in group D reduced the onset time for sensory block when compared to 0.75% ropivcaine with normal saline in group R.In the group R the onsetof sensory block was only 12.7±1.343 compared to group D which was shorter 9.97±0.928.This result was concurrent with the **Feroz Ahmad Dar et al** (2013) where they cocluded Sensory and motor block onset times were shorter in group RD(11.3± 2.61) minutes than in group R (13.12±2.30) minutes. This also correlated with study of **Y.N. Lin et al** where they concluded that the addition of dexmedetomidine to ropivacaine (Group D) shortened the sensory block onset time compared with the ropivacaine group (Group C) (95% confidence interval [CI] 4.18±5.26; $p < 0.05$).This finding also correlated with studies conducted by **Kaygusuz et al**.

Onset of motor block :

The study had shown that addition of 50 µg of dexmedetomidine to 25 ml of 0.75% ropivacaine in group D reduced the onset time for motor block when compared to 0.75% ropivcaine with normal saline in group R.In the group R the onset of motorblock was only 15.6±1.589 compared

to group D which was shorter 13.2±0.925. This results was correlated with following studies :

1) **Feroz Ahmad Dar et al** where they cocluded motor block onset times were shorter in group RD(15.61±4.37) minutes than in group R (13.40±3.73) minutes.

2) **Marhofer et al** observed that motor onset time was significantly faster in Group Rp (dexmedetomidine20 µg+ 0.75% ropivcaine) when compared with the other study groups [mean (SD)] [21 (15) vs 43 (25) min in Group RsD and 47 (36) min in Group R, P<0.05.

3) **K. Kaygusuz et al** this study aimed to investigate the effects of adding dexmedetomidine to levobupivacaine for an axillary brachial plexus block. They observed that Motor block onset time, in the levobupivacaine alone group is 15.75 (4.06) minutes and dexmedetomidine and levobupivacaine group is 14.25 (3.92) minutes. P(<0.05). They found a shorter onset time with dexmedetomidine concurrent with our study.

SENSORY BLOCK DURATION :

Our study shown that addition of 50 µg of dexmedetomidine to 25ml of 0.75% ropivacaine in group D prolongs the duration of sensory block when compared to 25 ml of ropivacaine 0.75% with normal saline

in group R. In group R the duration of sensory block was 419.6±9.665 minutes compared to group D which was 597.33±10.807 minutes.P (<0.05). This result was concurrent with

1) **Feroz Ahmad Dar et al** where they concluded that sensoray block duration is significantly greater with dexmedetomidine ropivacaine group (412.61□± 73.77) minutes than ropivacaine alone group(590.61□±80.30) minutes .P<0.0001.

2) **Malenfant Rancourt et al,** Compared the addition of dexmedetomidine with ropivacaine (RD) and ropivacaie alone (R)for tibial nerve block, they found that sensory block lasted longer in group RD than in group R(21.5 VS 16.2 hrs)p<0.001. This results was also correlated with the studies conducted by **D. Marhofer et al and Yu Zhang et a l.**

MOTOR BLOCK DURATION :

Our study had shown that addition of 50 µg of dexmedetomidine to 25 ml of 0.75% ropivacaine in group D prolongs the duration of motor block when compared to 25 ml of ropivacaine 0.75% with normal saline in group R but not in significant way. In group R the duration of motor block was 357.67±9.71 minutes compared to group D 360±8.3 minutes eventhough various studies decribes an significant increase in motor

block duration our study shows an insignificant increase in motor duration in dexmedetomidine group (RD) when compared to ropivacaine alone group(R).

RESCUE ANALGESIA :

Our study shown that addition of 50 µg of dexmedetomidine to 25 ml of 0.75% ropivacaine in group D prolongs the duration of analgesia and prolong the patients first analgesic request, 774.67 ±10.743 minutes, when compared to 25 ml of ropivacaine 0.75% with normal saline in group R 607.33±13.629. This result was correlated with following studies

1) **Feroz Dar et al** observed that duration of analgesia was significantly longer in group RD than in group R ($P< 0.001$) .They found that duration of analgesia was (600.14 ±90.82) minutes in ropivacaine dexmedetomidine group and (760.69±120.12) minutes in ropivacaine alone group.($P <0.0001$)
2) This result also correlated with the study **Kaygusuz et al** where they concluded that adding dexmedetomidine to axillary brachial plexus block increases the time to first analgesic use, and decreases total analgesic use with no side effects and also in studies conducted by Abdallah et al and swami et al .

HEMODYNAMIC STABILITY :

The hear rate, mean arterial pressure,remained stable both during intraoperative and postoperative period.The blood pressure never decreased below 20% of baseline values. No significant hypotension and bradycardia were observed and is correlated with the results of **Kaygusuz et al.**

SIDE -EFFECTS :

Side effects like bradycardia, hypotension, hypoxemia, nausea, vomiting, and any other side effects were not seen in any patients.

SUMMARY

This double blinded prospective randomized controlled study was done to evaluate the onset of sensory block,onset of motor blockade, duration of sensory and motor blockade, duration of analgesia and time for rescue analgesia and side effects of 50 µg of dexmedetomidine with 25 ml of 0.75% ropivacaine (Group D)vs 25 ml of 0.75% ropivacaine (Group R)given by axillary brachial plexus block approach for elective upperlimb forearm and hand surgeries. The onset of sensory and motor lock assessed by pin prick and bromage score respectively.The time of first rescue analgesics were assessed in the study.Hemodynamic parameters noted are mean arterial blood pressure,pulse rate ,and oxygen saturation.postoperative pain is assessed using visual analogue scale.If VAS 4and above rescue analgesics is administered. According to this study ,a significant shortening of sensory and motor block onset in group D ,when compared to group R.The addition of 50 µg of dexmedetomidine to 0.75% ropivacaine significantly prolonged the duration of analgesia. And the time for demand for analgesia was also prolonged. Sideeffects like bradycardia, hypotension, hypoxemia, nausea, vomiting, and any other side effects were not seen in any patients.The hemodynamic parameters are well maintained in both the groups. Thus the addition of dexmedetomidine to ropivacaine improve

the block quality,duration and provide better intraoperative conditions and better postoperative analgesia.

CONCLUSION

To conclude 50µg of dexmedetomidine seems to be a better adjuvant to 0.75% ropivacaine in axillary brachial plexus block in increasing the duration of analgesia, shortening the onset time of sensory and motor blockade and prolonging the duration of sensory blockade without sideeffects.

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STUDY PROFORMA

NAME :

IP.NO: ASA ;

AGE & SEX:

WEIGHT:

DIAGNOSIS: PROCEDURE:

Co Morbid illness:

Personal History:

H/O Allergy:

GENERAL EXAMINATION:

Built & Nourishment

Pallor /Cyanosis /Clubbing /Icterus /pedal edema

PR /min NIBP mm/Hg RR /min

Airway examination: MPC Neck movements

Systemic examination: CVS RS P/A CNS

INVESTIGATIONS :

TIME	EVENTS	PR/MIN	NIBP mm/Hg(MAP)	Sp02%	IV FLUIDS	SIDE EFFECTS
O min						
5 min						
1O min						
15 min						
20 min						
25 min						
30 min						
1 hour						
2 hours						
3 hours						
4 hours						
5 hours						
6 hours						
7 hours						
8 hours						
9 hours						

PARAMETERS	Group D	Group R
Onset sensory (Minutes)		
Onset motor (Minutes)		
Duration sensory (Minutes)		
Duration motor (minutes)		
Rescue analgesia (minutes)		
VAS at recovery		

GROUP D MASTER CHART

S. No	Name	Age	wt in kg	Proceedure	ASA	Onset sensory (min)	Onset motor (min)	Sensory duration (min)	Motor duration (min)	Rescue analgesia (min)	VAS at recovery
1	Duraisamy	56	72	Neurofibroma rt distal forerm excision	ASA 2	11	13	610	490	780	3
2	Krishnamoorthy	47	70	Fracture distal radius ORIF	ASA 2	11	14	600	470	770	3
3	Pradeep	19	66	Fracture distal radius ORIF	ASA1	11	13	590	470	760	3
4	Gopi krishnan	18	68	Post traumatic raw area lt wrist flap cover	ASA1	10	14	600	480	770	3
5	Vignesh	28	72	dupytrene's contracture release	ASA1	11	13	590	470	760	3
6	Syed abdullah	24	68	Fracture distal radius ORIF	ASA1	10	13	600	500	780	3
7	Amsa	55	68	Fracture distal radius ORIF	ASA2	12	15	590	460	760	3
8	Prabhu	28	69	Fracture distal radius ORIF	ASA 1	10	14	580	470	750	4
9	Periyasamy	50	72	Osteomyleitis distal radius sequestrectomy	ASA2	10	12	600	480	770	3
10	Anbu	37	68	LT ulna distal 1/3 fracture ORIF	ASA1	9	15	610	480	790	3
11	Kannan	25	66	Fracture distal radius ORIF	ASA1	10	12	600	475	770	4
12	Moorthi	49	70	cellulitis rt hand wound debridement	ASA2	8	12	580	490	790	3
13	Jeyaraman	25	66	traumatic amputation lt little finger stump closure	ASA1	9	14	620	460	780	4
14	Saravanan	19	70	Fracture distal radius ORIF	ASA1	10	12	580	480	760	3
15	Kannan	24	73	Fracture distal radius ORIF	ASA1	8	12	590	490	790	3
16	Krishnav	50	68	Fracture distal radius ORIF	AS	10	13	600	470	780	3

S. No	Name	Age	wt in kg	Proceedure	ASA	Onset sensory (min)	Onset motor (min)	Sensory duration (min)	Motor duration (min)	Rescue analgesia (min)	VAS at recovery
	el				A2						
17	Sakunthala	54	68	Fracture distal radius ORIF	ASA2	10	14	610	490	770	3
18	Anbu	31	70	Fracture distal radius ORIF	ASA1	11	13	590	470	780	3
19	Murugan	40	68	Fracture distal radius ORIF	ASA2	10	14	590	470	780	3
20	Mohanapriya	28	70	lt extensor tendon reduction	ASA1	9	13	600	480	790	3
21	Narayanasamy	49	68	Fracture distal radius ORIF	ASA2	9	12	610	480	780	3
22	Andal	50	68	Fracture distal radius ORIF	ASA2	10	13	590	490	780	3
23	Subramani	63	66	2nd metacarpal fracture rt k wire fixation	ASA2	11	13	590	475	770	3
24	Murugan	36	70	Fracture distal radius ORIF	ASA1	9	12	600	470	790	3
25	Pottyammal	60	66	Fracture distal radius ORIF	ASA2	10	12	610	480	780	3
26	Ravi	45	67	Fracture distal radius ORIF	ASA2	10	14	600	490	770	3
27	Raja	34	68	Fracture distal radius ORIF	ASA 1	11	14	590	470	760	3
28	Suresh	23	67	Fracture distal radius ORIF	ASA1	9	13	580	480	770	3
29	Arshad	24	66	Fracture distal radius ORIF	ASA1	10	14	610	490	780	3
30	Roshan	19	69	Fracture distal radius ORIF	ASA1	10	14	610	490	780	3

GROUP R MASTER CHART

S. No	Name	Sex	Wt	Procedure	Asa-ps	Onset of sensory block (min)	Onset motor block (min)	Sensory duration (min)	Motor duration (min)	Rescue analgesia (min)	VAS
1	Selvi	35	66	Postburn raw area lower forearm flap cover	ASA 1	13	15	412 min	350 min	600 min	4
2	Arumugam	58	64	EDL Cut injury tendon repair	ASA 2	12	14	420 min	360 min	620 min	4
3	Satya	35	68	Cut EDL tendon repair tendon repair	ASA 1	14	16	420 min	370 min	590 min	5
4	Harikrishnan	18	64	Post traumatic raw area thumb flap cover	ASA 1	12	14	415 min	355 min	610 min	4
5	Selvaraj	55	70	Rt index extensor division repair	ASA 2	14	18	425 min	370 min	610 min	5
6	Anbu	31	68	Post traumatic raw area wound debride ment	ASA 1	16	20	420 min	350 min	600 min	4
7	Muthuraj	49	70	EDL tendon injury tendon repair	ASA 2	13	15	440 min	360 min	580 min	5
8	Dinesh	21	68	Extensor tendon injury exploration	ASA 1	13	15	414 min	350 min	630 min	5
9	Veerabathran	32	72	Fracture distal radius ORIF	ASA 1	12	14	410 min	340 min	610 min	4
10	Nithyanandan	22	70	Post raw area rt hand ssg	ASA 1	11	13	410 min	340 min	590 min	4
11	Ponpandy	40	68	Claw hand secondary repair	ASA 2	12	16	415 min	360 min	610 min	4
12	Ponammal	56	68	Post traumatic raw area ssg	ASA 2	10	14	410 min	360 min	620 min	4
13	Sivakami	30	60	Lt thumb injury flap cover	ASA 1	14	18	430 min	340 min	580 min	4
14	Murugan	39	72	Fracture distal radius ORIF	ASA 1	13	15	420 min	370 min	620 min	4
15	Arumugam	45	68	Fracture distal radius ORIF	ASA 2	13	15	425 min	360 min	610 min	4
16	Bhupendr	50	68	Post traumatic area index	ASA	14	16	450	360	610	5

S. No	Name	Sex	Wt	Procedure	Asa-ps	Onset of sensory block (min)	Onset motor block (min)	Sensory duration (min)	Motor duration (min)	Rescue analgesia (min)	VAS
	an			finger ssg	2			min	min	min	
17	Nandhini	19	65	Post burn contracture ring finger release	ASA 1	13	15	412 min	370 min	600 min	5
18	Manoharan	27	70	Grade 2 lt middle finger osteoma excision	ASA 1	10	14	410 min	340 min	580 min	4
19	Rajkumar	30	66	Fracture distal radius ORIF	ASA 1	13	16	420 min	350 min	600 min	4
20	Selvaraj	38	64	Fracture 5th metacarpal closed reduction	ASA 1	14	15	410 min	360 min	630 min	4
21	Yasodha	48	65	Fracture distal radius ORIF	ASA 2	12	16	430 min	370 min	620 min	4
22	Ravi	32	66	Fracture distal radius ORIF	ASA 1	11	15	430 min	350 min	610 min	4
23	Ajith	22	68	Fracture distal radius ORIF	ASA 1	14	14	420 min	360 min	620 min	4
24	Arul	27	67	Fracture distal radius ORIF	ASA 1	12	16	410 min	370 min	610 min	4
25	Ramesh	45	70	Fracture distal radius ORIF	ASA 2	12	14	415 min	360 min	600 min	4
26	Prabhu	28	66	Fracture distal radius ORIF	ASA 1	13	16	430 min	370 min	620 min	4
27	Poongavanam	40	68	Fracture distal radius ORIF	ASA 1	14	18	420 min	360 min	620 min	4
28	Saravanan	24	70	Fracture distal radius ORIF	ASA 1	12	17	410 min	350 min	610 min	4
29	Seeman	50	68	Fracture distal radius ORIF	ASA 2	11	16	420 min	360 min	610 min	4
30	Ramesh	40	68	Fracture 4th metacarpal k wire fixation	ASA 2	14	18	415 min	350 min	600 min	4

GROUP R MEAN ARTERIAL PRESSURE MASTER CHART

	min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 min	420 min	480 min	540 min	600 min	660 min	720
	)6	108	104	106	109	109	108	110	108	108	107	110	110	109	109	108	10
	)0	98	98	97	99	99	99	99	101	100	101	101	100	100	101	102	10
	)4	102	103	103	102	102	101	101	104	105	105	105	104	104	104	103	10
	)8	107	107	10	108	107	109	109	108	108	107	107	108	109	109	108	10
	)7	108	108	109	109	106	106	107	107	108	108	108	107	107	106	106	10
	)3	103	104	102	101	101	102	102	103	103	102	102	102	101	101	101	10
	)2	103	102	104	103	102	103	102	103	102	104	101	102	103	102	102	10
	)8	108	108	107	107	107	107	108	108	107	107	106	107	106	107	108	10
	)6	105	106	105	106	104	105	104	104	105	105	106	106	107	105	106	10
	)6	107	107	106	106	106	107	107	107	106	106	106	107	107	106	106	10
	)4	102	103	103	102	102	101	101	104	105	105	105	104	104	104	103	10
	)8	107	107	10	108	107	109	109	108	108	107	107	108	109	109	108	10
	)7	108	108	109	109	106	106	107	107	108	108	108	107	107	106	106	10
	)3	103	104	102	101	101	102	102	103	103	102	102	102	101	101	101	10
	)2	103	102	104	103	102	103	102	103	102	104	101	102	103	102	102	10
	)8	108	108	107	107	107	107	108	108	107	107	106	107	106	107	108	10
	)6	105	106	105	106	104	105	104	104	105	105	106	106	107	105	106	10
	)6	107	107	106	106	106	107	107	107	106	106	106	107	107	106	106	10
	8	97	99	99	99	99	101	100	101	101	101	101	100	100	101	102	10
	)3	103	102	102	101	101	104	105	105	105	105	105	104	104	104	103	10
	)7	10	108	107	109	109	108	108	107	107	107	107	108	109	109	108	10
	)8	109	109	106	106	107	107	108	108	108	108	108	107	107	106	106	10
	)4	102	101	101	102	102	103	103	102	102	102	102	102	101	101	101	10
	)2	104	103	102	103	102	103	102	104	101	104	101	102	103	102	102	10
	)8	107	107	107	107	108	108	107	107	106	107	106	107	106	107	108	10

	min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 min	420 min	480 min	540 min	600 min	660 min	720
	)6	105	106	104	105	104	104	105	105	106	105	106	106	107	105	106	10
	)7	106	106	106	107	107	107	106	106	106	106	106	107	107	106	106	10
	)3	103	102	102	101	101	104	105	105	105	105	105	104	104	104	103	10
	)7	10	108	107	109	109	108	108	107	107	107	107	108	109	109	108	10
	)8	109	109	106	106	107	107	108	108	108	108	108	107	107	106	106	10

GROUP D MEAN ARTERIAL PRESSURE MASTER CHART

S. No	0 min	5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360
1	109	108	108	107	107	10	108	107	109	109	108	108	108
2	108	107	107	108	108	109	109	106	106	107	107	109	109
3	102	102	103	103	104	102	101	101	102	102	103	102	101
4	103	103	102	103	102	104	103	102	103	102	103	104	103
5	108	108	108	108	108	107	107	107	107	108	108	107	107
6	106	106	106	105	106	105	106	104	105	104	104	105	106
7	107	106	106	107	107	106	106	106	107	107	107	106	106
8	104	103	104	102	103	103	102	102	101	101	104	103	102
9	109	108	108	107	107	10	108	107	109	109	108	107	108
10	108	107	107	108	108	109	109	106	106	107	107	109	109
11	102	102	103	103	104	102	101	101	102	102	103	102	101
12	103	103	102	103	102	104	103	102	103	102	103	104	103
13	108	108	108	108	108	107	107	107	107	108	108	107	107
14	106	106	106	105	106	105	106	104	105	104	104	105	106
15	107	106	106	107	107	106	106	106	107	107	107	106	106

S. No	0 min	5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360
16	100	100	100	102	101	102	101	102	101	100	101	102	102
17	104	102	103	103	102	102	102	103	104	105	105	103	103
18	108	107	107	108	108	107	109	109	108	108	107	107	107
19	107	108	108	109	109	106	106	107	107	108	108	107	108
20	103	103	104	102	101	101	102	102	103	103	102	103	102
21	104	103	104	102	103	103	102	102	101	101	104	105	105
22	109	108	108	107	107	10	108	107	109	109	108	108	107
23	108	107	107	108	108	109	109	106	106	107	107	108	108
24	102	102	103	103	104	102	101	101	102	102	103	103	102
25	103	103	102	103	102	104	103	102	103	102	103	102	104
26	108	108	108	108	108	107	107	107	107	108	108	107	107
27	106	106	106	105	106	105	106	104	105	104	104	105	105
28	107	106	106	107	107	106	106	106	107	107	107	106	106
29	104	103	104	102	103	103	102	102	101	101	104	105	105
30	109	108	108	107	107	10	108	107	109	109	108	108	107

GROUP R MEAN PULSE RATE MASTER CHART

S.No	0 min	5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 m
1	84	82	84	82	82	84	84	84	82	84	86	84	84
2	84	82	84	82	84	82	82	84	84	82	84	84	82
3	84	84	82	86	84	84	82	82	82	82	82	82	82
4	82	80	80	80	80	80	82	82	84	82	82	82	84
5	82	80	86	84	84	82	80	80	78	78	78	78	80
6	84	82	84	82	82	84	80	80	78	78	78	78	80
7	82	84	84	86	82	82	82	82	82	80	80	78	78
8	76	78	80	80	78	76	78	76	78	80	82	80	80
9	82	82	80	80	82	82	80	80	80	80	82	82	82
10	84	82	80	82	78	76	78	80	82	82	80	80	80
11	82	84	82	82	84	84	84	82	84	82	84	84	82
12	82	82	84	82	82	84	84	84	82	84	86	84	84
13	82	82	84	82	84	82	82	84	84	82	84	84	82
14	84	84	82	86	84	84	82	82	82	82	82	82	82
15	80	80	80	80	80	80	82	82	84	82	82	82	84
16	80	80	86	84	84	82	80	80	78	78	78	78	80
17	82	82	82	82	82	80	80	78	84	82	84	82	84
18	78	76	78	76	78	80	82	80	84	82	84	84	82
19	82	82	80	80	80	80	82	82	82	82	82	82	84
20	78	76	78	80	82	82	80	80	84	78	76	76	78
21	84	84	84	82	84	82	84	84	84	83	83	82	82
22	82	84	84	84	82	84	86	84	80	82	84	84	82
23	84	82	82	84	84	82	84	84	84	82	82	82	80
24	84	84	82	82	82	82	82	82	82	84	82	82	82
25	80	80	82	82	84	82	82	82	82	82	84	84	82
26	84	82	80	80	78	78	78	78	80	80	82	84	82
27	82	84	80	80	78	78	78	78	80	82	82	84	84

S.No	0 min	5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 m
28	82	82	82	82	82	80	80	78	82	82	82	82	80
29	78	76	78	76	78	80	82	80	82	80	82	82	80
30	82	84	84	84	82	82	84	84	82	84	84	82	84

GROUP D MEAN PULSE RATE MASTER CHART

S. No	0 min	5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 min	42
1	88	86	84	84	84	82	82	84	84	82	84	82	82	
2	86	86	84	82	82	80	80	82	84	82	84	82	84	
3	84	84	84	82	84	84	82	82	84	84	82	86	84	
4	80	82	84	86	82	82	82	82	82	80	80	80	80	
5	78	80	82	78	76	78	80	82	82	80	86	84	84	
6	78	80	82	78	76	78	82	84	84	82	84	82	82	
7	74	80	80	82	82	84	82	82	82	84	84	86	82	
8	76	78	80	78	78	76	78	76	76	78	80	80	78	
9	78	78	80	82	84	84	83	83	82	82	80	80	82	
10	80	82	84	82	84	82	82	84	84	82	80	82	78	
11	84	88	86	84	84	84	82	82	82	84	82	82	84	
12	86	84	84	84	82	82	84	84	82	82	84	82	82	
13	86	84	82	82	80	80	82	84	82	82	84	82	84	
14	84	84	82	84	84	82	82	84	84	84	82	86	84	
15	82	84	86	82	82	82	82	82	80	80	80	80	80	
16	80	82	78	76	78	80	82	82	80	80	86	84	84	
17	80	82	78	76	78	82	84	84	82	82	84	82	82	
18	80	80	82	82	84	82	82	82	84	84	84	86	82	
19	78	80	78	78	76	78	76	76	78	78	80	80	78	
20	78	80	82	84	84	83	83	82	82	82	80	80	82	
21	82	84	82	84	82	82	84	84	82	82	80	82	78	
22	80	84	86	82	82	82	82	82	80	84	82	82	84	
23	84	88	86	84	84	84	82	82	82	84	82	82	84	
24	86	84	84	84	82	82	84	84	82	82	84	82	82	
25	82	84	82	82	80	80	82	84	82	82	84	82	84	
26	86	84	82	84	84	82	82	84	84	84	82	86	84	
27	86	84	86	82	82	82	82	82	80	80	80	80	80	

S. No	0 min	5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 min	42
28	88	82	78	76	78	80	82	82	80	80	86	84	84	
29	84	82	78	76	78	82	84	84	82	82	84	82	82	
30	82	84	84	84	82	82	84	84	82	82	84	82	82	

GROUP R MEAN OXYGEN SATURATION (SPO2) MASTER CHART

S.No	0 min	5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 min	420 min	480 min	540 min	600
1	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100
2	100%	100%	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	99%	99%	99%	99%	99
3	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100
4	100%	100%	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	100%	100%	100%	100%	100
5	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	99%	99
6	100%	100%	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	99%	99%	99%	99%	99
7	99%	99%	99%	99%	99%	99%	99%	99%	99%	100%	100%	100%	99%	99%	99%	99%	99
8	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100
9	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100
10	100%	100%	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	100%	100%	100%	100%	100
11	99%	100%	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	99%	99%	99%	99%	99
12	100%	99%	99%	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	100%	100
13	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99
14	100%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99
15	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100
16	99%	100%	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	99%	99%	99%	99%	99
17	99%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100
18	99%	99%	99%	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	100%	100
19	100%	100%	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	99%	99%	99%	99%	99
20	100%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99
21	100%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99
22	99%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100
23	100%	100%	100%	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	100%	100
24	99%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100
25	99%	100%	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	99%	99%	99%	99%	99
26	100%	100%	99%	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	100%	100
27	100%	100%	100%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99

S.No	0 min	5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 min	420 min	480 min	540 min	600
28	99%	100%	100%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99
29	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%	99
30	100%	100%	99%	100%	100%	100%	99%	100%	100%	100%	100%	100%	100%	100%	100%	100%	99

GROUP D MEAN OXYGEN SATURATION (SPO2)
MASTER CHART

	5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 min	420 min	480 min	540 min	600 min	660 min	720 min	780 m
	100%	100%	100%	100%	100%	100%	100%	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%
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		5 min	10 min	15 min	20 min	25 min	30 min	60 min	120 min	180 min	240 min	300 min	360 min	420 min	480 min	540 min	600 min	660 min	720 min	780 m
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